

FRCEM INTERMEDIATE

**A LITERATURE-GUIDED APPROACH TO
THE DIAGNOSIS AND MANAGEMENT
OF EMERGENCY CONDITIONS
ACCORDING TO THE 2015 FRCEM
INTERMEDIATE CURRICULUM**

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FRCEM Intermediate: Short Answer Question

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PREFACE

The FRCEM Intermediate SAQ is a direct replacement for the MRCEM Part B examination and continues to be mapped to the competences of Year 1-3 of the Emergency Medicine 2015 Curriculum which is available on the **2015 Curriculum page** of the RCEM website.

The Short Answer Question Paper comprises of a 3-hour paper of 60, 3 mark questions.

There is a limited amount of materials and resources available since the changes made to the curriculum.

This book has been written in response to suggestions from colleagues who have asked for compiled Notes on the FRCEM intermediate exam adapted and mapped more particularly to the newly revised college Curriculum.

Readers will realize and understand that materials found in this book are slightly more progressive than some usual books we use to when preparing for the MCEM part B exams.

The book is organized in sections in accordance with the Curriculum, helping candidates to follow their progress and organize their preparation.

A dedicated section on **"Practical Procedures"** (36 marks) along with **"Common competences"** (21 marks) have been inserted to help candidates achieve higher score and improve their passing rate. Numerous images are also included to facilitate the candidates while reading.

Various Chapters have been infused with clinical scenario questions and answers to facilitate assimilation of the topic.

This is the only book covering the curriculum from A to Z with the emergency management approach for every condition.

The principles in this book may be applied while preparing for the FRCEM SJP exams and to our daily practice as Emergency Doctors.

All the best for your exam!

Dr Tatum Ronne

ACKNOWLEDGMENTS

You may find it peculiar that such big book is written by a single author. I can simply tell you that's not the case. Many of my colleagues have supported me and helped me see this book through to completion.

Many guidelines presented in this book originated from:

- Royal College of Emergency Medicine (www.rcem.ac.uk),
- National Institute for Health and Care Excellence (www.nice.org.uk),
- British Thoracic Society (www.brit-thoracic.org.uk),
- Resuscitation Council UK (www.resus.org.uk),
- American Heart Association (www.heart.org),
- Toxbase (www.toxbase.org),
- Life in the fast lane (www.lifeinthefastlane.com)

I owe my dedicated work to the above organizations.

Although these sites have provided most of my data, I had to spend an entire year reading, compiling, editing and organizing all the necessary information to accommodate essential topics and guidelines. The aim of that hard work was to match the 2015 FRCM Intermediate Curriculum published by the Royal College of Emergency Medicine.

Many times, I felt like giving up and focusing solely on existing materials when preparing for my exams. Yet each time an inner voice, doubled by my wife's support, overcame my laziness and urged me to pursue my ultimate dream, being the first to write a book completely matching the current curriculum.

As with the FRCM Primary, my thanks and gratitude to my best friend and wonderful wife **Marlene Katoy Issa**, to my lovely children **Tatiana Issa**, **Kevin Issa** and **Ryan Moussa Issa Jr.** Their friendship, support, and encouragements are most meaningful.

I was fortunate enough to have a group of excellent colleagues and consultants who believed in my power to start and complete a project. They are always around for any help, idea and support.

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MAJOR & ACUTE PRESENTATIONS

18 QUESTIONS

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ED Registrar**

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CHAPTER 1. ALLERGIC REACTIONS

1. ANAPHYLAXIS

• DEFINITION

- Anaphylaxis is a severe, life-threatening, systemic hypersensitivity reaction, typically of rapid onset; associated with Airway, breathing and/or circulatory involvement.

• Anaphylaxis is likely when all three of the following criteria are met:

- *Acute onset of illness and sudden progression*
- *Skin and/or mucosal changes, e.g. flushing, urticaria, angioedema*
- *Life threatening Airway and/ or Breathing and/ or Circulation problems*

• *Skin or mucosal changes alone are not a sign of an anaphylactic reaction. Skin or mucosal changes can be subtle or absent in up to 20% of reactions, e.g. some patients can have only a decrease in blood pressure, i.e. a circulation problem.*

• Pathophysiology

- Anaphylaxis can be caused by an either allergic or non-allergic mechanism.
- The clinical presentation and management is the same regardless of whether the reaction has an allergic or nonallergic mechanism.
- Allergic anaphylaxis is an example of **immediate type 1 hypersensitivity**.
- The response is caused by the binding of an antigen to an antigen-specific antibody leading to mediating mast cell activation. Histamine and other mediators, including leukotrienes, tumour necrosis factor and various cytokines, are released from mast cells and basophils following exposure to this antigen. This causes bronchial smooth muscle tone to increase (causing wheeze and shortness of breath), decreased vascular tone and increased capillary permeability (leading to hypotension and an urticarial rash). The response is usually **uniphasic**, although a **biphasic response** occurs in approximately 20% of individuals.

• COMMON AGENTS KNOWN TO CAUSE ANAPHYLAXIS INCLUDE:

- **Drugs:**
 - **Antibiotics:** Penicillin is the most common cause of drug induced anaphylaxis,
 - **Aspirin and NSAIDs:** second most common cause of drug induced anaphylaxis.
 - **Angiotensin Converting Enzyme Inhibitors**
- **Food:** e.g. peanuts, egg and seafood (food is the most common cause of anaphylaxis in children). The clinical cross-reactivity with other foods in the same group is unpredictable.
- **Insect stings:** bees and wasps
- **Hereditary C1 esterase inhibitor deficiency:** usually inherited as an autosomal dominant, but also occurs with lymphoma and certain connective tissue disorders.
- **Idiopathic**

• Less commonly:

- Physical triggers, e.g. exercise, cold
- Biological fluids, e.g. transfusions, semen
- Latex

• SIGNS AND SYMPTOMS

- **Skin and mucosal:** urticaria, erythema, pruritus
- **Airway problems:** lip and tongue swelling/ angioedema, nasal congestion, sneezing, tightness of throat/ hoarse voice/ stridor

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- o **Breathing problems:** tachypnoea, bronchospasm/ wheeze, increased mucous secretions, exhaustion, confusion, cyanosis, respiratory arrest.
- o **Circulation problems:** hypotension, tachycardia, arrhythmia, myocardial ischemia, cardiac arrest.
- o **Neurological problems:** confusion, agitation, loss of consciousness.
- o **Gastrointestinal:** stomach cramps, nausea, vomiting, diarrhoea
- o **Other:** feeling of impending doom
- **INVESTIGATION**
 - o **Mast cell tryptase** is released during the anaphylactic reaction and may be measured in the blood. It reaches its peak blood concentration approximately **1-2 hours** after the reaction. This is useful to aid later diagnosis and treatment and can help in the diagnosis in uncertain cases. The half-life of tryptase is short (approximately 2 hours), and concentrations may be back to normal **within 6-8 hours**, so timing of any blood samples is very important.
 - o **Three timed samples:**
 - **Initial sample as soon as feasible** after resuscitation has started – do not delay resuscitation to take sample.
 - **Second sample at 1-2 hours after** the start of the symptoms.
 - **Third sample either at 24 hours or in convalescence** (for example in a follow up allergy clinic). This provides baseline tryptase levels – some individuals have an elevated baseline level.
- **TREATMENT OF ANAPHYLAXIS**
 - o **Epinephrine** is the most important drug in the treatment of anaphylaxis.
 - o **Oxygen and fluid resuscitation**
 - o **Antihistamines:**
 - o **H1 blockers** help to overcome the histamine-induced vasodilatation.
 - o **Corticosteroids** are slow acting drugs that take between six and eight hours to reduce the immune-mediated reaction. They may be useful in preventing, or reducing the severity of, a biphasic response.
 - *Anaphylaxis due to C1 esterase inhibitor deficiency is resistant to adrenaline, steroids and antihistamines and needs treatment with **C1 esterase inhibitor concentrate or fresh frozen plasma***
- **FURTHER MANAGEMENT**
 - o Most patients who have suffered an anaphylactic reaction will need admission and observation **for 6 hours**.
 - o Patients with the following may need observation for **up to 24 hours**:
 - *Previous history of biphasic reactions or known asthmatics*
 - *Possibility of continuing absorption of allergen (fully eaten peanut butter sandwich)*
 - *Poor access to emergency care*
 - *Presentation in the evening or at night*
 - *Severe reactions with slow onset caused by idiopathic anaphylaxis.*
 - o **Biphasic reactions** are not easy to predict. Patients who have suffered an anaphylactic reaction are likely to suffer future episodes and follow-up should be arranged.
 - o **Outpatient follow-up** is useful to help identify the allergen and provide training in the use of an **epipen**.
 - o Patients should be given an **epipen** and instructions as to how to use it.
 - o There is no benefit from providing an additional course of steroids.

ANAPHYLAXIS ALGORITHM

Anaphylactic Reaction?

Airway. Breathing, Circulation, Disability, Exposure?

Diagnosis- look for:

- Acute onset of illness
- Life-threatening Airway and/or Breathing and/or circulation problems
- And usually skin changes

- Call for help
- Lie the patient flat
- Raise patient's legs

ADRENALINE²

When skills and equipment available:

- Establish Airway
- High flow oxygen
- IV fluid challenge
- Chlorphenamine
- Hydrocortisone

Monitor:

- Pulse oximetry
- ECG
- Blood Pressure

1. Life-threatening problems:

- **Airway:** intraoral swelling, hoarseness, stridor, swollen tongue
- **Breathing:** rapid breathing, wheeze, fatigue, cyanosis, SPO₂ <92%, confusion
- **Circulation:** pale, clammy, low BP, faintness, drowsy/ coma

2. Adrenaline (give IM unless experienced with IV adrenaline)

IM dose of 1:1000 Adrenaline (repeat after 5min if no better)

- Adult: 500 mcg IM (0.5 ml)
- Child > 12 years: 500 mcg IM (0.5 ml)
- Child 6-12 years: 300 mcg IM (0.3 ml)
- Child < 6 years: 150 mcg IM (0.15 ml)

Adrenaline IV only if experienced specialists

Titrate: Adults 50 mcg, Children 1mcg/Kg

3. IV Fluid challenge:

- Adult: 500-1000 ml
- Child: crystalloid 20ml/Kg

Stop IV colloid if this might be the cause of anaphylaxis

Age	4. Chlorphenamine (IM/ slow IV)	5. Hydrocortisone (IM/ slow IV)
Adult	• 10 mg	• 200 mg
Child > 12 years	• 5 mg	• 100 mg
Child 6-12 years	• 2.5 mg	• 50 mg
Child < 6 years	• 250mcg/Kg	• 25 mg

2. URTICARIA (HIVES)

- Histamine mediated **localised oedema of the dermis**. It is at one end of the allergic reaction spectrum with anaphylactic shock at the other end.
- Exposure to an allergenic protein produces IgE mediated mast cell degranulation and histamine release.
- This produces vascular dilation and transudation of fluid from the affected vessels.
- *Unlike in allergic angioedema and anaphylaxis, this vascular dilatation is limited to the dermis.*

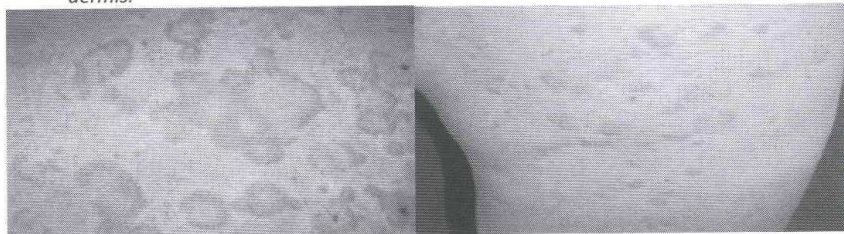


Figure 1.1.2. Urticaria (Hives)

3. ANGIOEDEMA (AE)

- Pathogenetically similar to urticaria but involves the **deeper dermal and subcutaneous tissue**. The aetiology of angioedema can be either **allergic** (IgE and histamine mediated as in urticaria) or **non-allergic**.
- **90%** of attacks of angioedema are of the allergic type.
- Non-allergic AE can be further subdivided into:
 - **Drug induced** (e.g. ACE-inhibitors),
 - **Hereditary** (C1-esterase-inhibitor deficiency),
 - **Acquired, Idiopathic and Pseudoallergenic**.
- Unlike the allergic form, the non-allergic drug induced, hereditary and acquired forms are mediated by bradykinin.
- The commonest drug classes associated with angioedema are listed in the table below:

Top 10 Drugs / Drug classes associated with angioedema	
1. ACE inhibitors	6. SSRIs
2. Angiotensin 2 antagonists	7. Other antidepressants
3. Vaccines	8. Bupropion
4. NSAIDS	9. Statins
5. COX-II Inhibitors	10. Proton Pump Inhibitors

4. HEREDITARY ANGIOEDEMA (HAE)

- HAE is an autosomal dominant condition caused by **C1 esterase inhibitor deficiency or functional deficiency**.
- This can be confirmed clinically by low levels of **C4 and C1 esterase inhibitor function**.

SIGNS & SYMPTOMS

- Urticaria produces **wheals and papules** of non-pitting, oedematous, erythematous and intensely pruritic skin. These can appear as crops mainly on the limbs and trunk and can spontaneously resolve quickly.

- **Angioedema** involves the deeper dermal structures with little pruritis. It commonly involves swelling of the face, lips, mouth, tongue, extremities and the genitalia in men.
- Laryngeal involvement can produce stridor and lead to complete airway obstruction.
- Angioedema is also associated with abdominal symptoms caused by bowel wall oedema, such as colic-like pain, nausea, vomiting and diarrhoea.
- However cutaneous attacks are the most common form.
- *Symptoms can occur singly or in combination; acute urticaria presents with angioedema in about 50% of cases and alone in 40% of cases.*
- *Angioedema presents alone in approximately 10% of cases which should prompt consideration of a non-allergic form.*



Figure 1.1.3. Angioedema of the lips and hands

ALLERGIC ANGIOEDEMA	NON-ALLERGIC ANGIOEDEMA (CEID)	ANAPHYLAXIS
Anatomically localised attack	Anatomically localised attack	Systemic symptoms
Urticaria	Gradual onset	Rapid onset and progression
Pruritis	No Pruritis	Respiratory failure (wheeze, fatigue, cyanosis, hypoxia, tachypnoea)
Normotension	Previous identical episodes	Cardiovascular Collapse (Diaphoretic, hypotensive, tachycardia, drowsiness)
	Abdominal pain	
	Normotension	

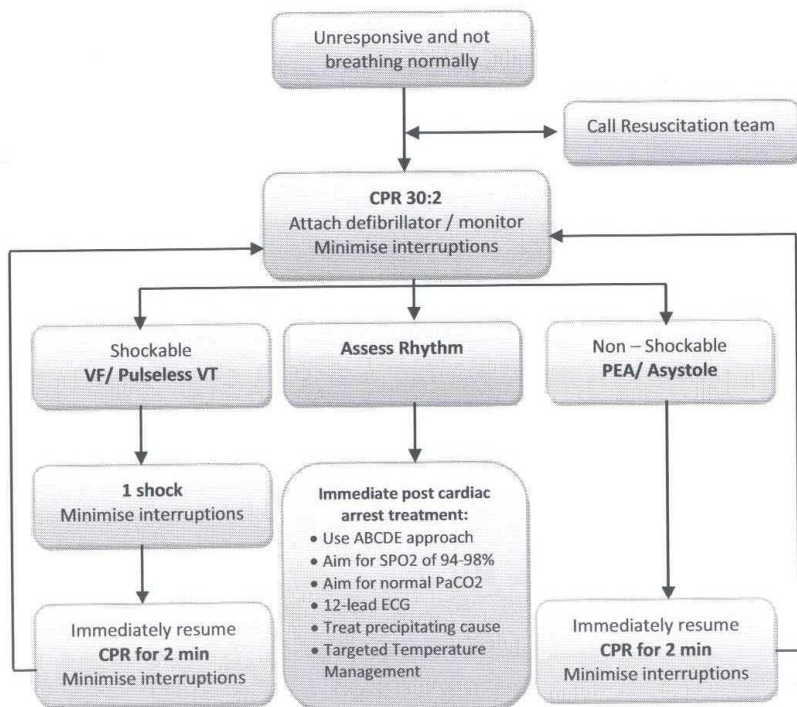
- **DIFFERENTIAL DIAGNOSIS**
 - Evolving Anaphylaxis
 - Cellulitis
 - Erysipelas
 - Lymphoedema
 - SLE and Contact Dermatitis

5. SCOMBROID POISONING

- **Scombroid poisoning** is a disease due to the ingestion of contaminated food (mainly fish).
- In scombroid poisoning, bacteria have grown during improper storage of the dark meat of the fish and the bacteria produce scombroid toxin. Scombroid toxin, or poison, is probably a combination of histamine and histamine-like chemicals.
- The toxin or poison does not affect everyone who ingests it.

CHAPTER 2. CARDIO-RESPIRATORY ARREST**A. ADVANCED CARDIAC LIFE SUPPORT**

- o Routine cricoid pressure not recommended
- o Use continuous capnography if intubated
- o Emphasis on high quality CPR
- o **Atropine** no longer used in PEA/Asystole
- o **Adenosine** is recommended in stable, undifferentiated, regular monomorphic wide complex tachycardia
- o Trial of chronotropic drugs before pacing suggested for unstable bradycardia



During CPR:	Treat Reversible causes:	Consider:
<ul style="list-style-type: none"> • Ensure high quality Chest compressions • Minimise interruptions to compressions • Give Oxygen • Use Waveform capnography • Continuous compressions when advanced airway in place • Vascular access (IV/ IO) • Give Adrenaline every 3-5 min • Give Amiodarone after 3 shocks 	<ul style="list-style-type: none"> • Hypoxia • Hypovolaemia • Hypo/ Hyperkalaemia/Metabolic • Hypothermia • Thrombosis: Coronary/pulmonary • Tension pneumothorax • Tamponade- cardiac • Toxins 	<ul style="list-style-type: none"> • Ultrasound imaging • Mechanical chest compressions to facilitate transfer/ treatment • Coronary angiography and percutaneous coronary intervention • Extracorporeal CPR

Figure 1.2.1. Adult advanced life support algorithm

REVERSIBLE CAUSES

The "Hs"	The "Ts"
<ul style="list-style-type: none"> o Hypoxia o Hypovolaemia o Hypokalaemia/Hyperkalaemia, o Hypothermia o Hydrogen: Acidaemia o Other metabolic disorders: <ul style="list-style-type: none"> o Hypoglycaemia, o Hypocalcaemia, 	<ul style="list-style-type: none"> o Thrombosis: coronary or pulmonary o Tension pneumothorax o Tamponade – cardiac o Toxins

- **Hypoxia:**
 - o Adequate ventilation with the maximal possible inspired oxygen during CPR.
 - o Adequate chest rise and bilateral breath sounds.
 - o Check that the tracheal tube is not misplaced in a bronchus or the oesophagus.
- **Hypovolaemia:**
 - o Usually due to severe haemorrhage >>> **Stop the haemorrhage**
 - o Restore intravascular volume with fluid and blood products.
- **Hyperkalaemia, Hypokalaemia, Hypocalcaemia, Acidaemia and Other Metabolic Disorders:**
 - o Detected by biochemical tests or suggested by the patient's medical history (e.g. renal failure).
 - o Give **IV calcium chloride** in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.
- **Hypothermia:**
 - o Should be suspected based on the history such as cardiac arrest associated with drowning.
 - o Rewarm the patient up to 34°C
- **Coronary thrombosis:**
 - o Associated with an **acute coronary syndrome** or **ischaemic heart disease** is the most common cause of sudden cardiac arrest.
 - o An ACS is usually diagnosed and treated after ROSC is achieved.
 - o If an ACS is suspected, and ROSC has not been achieved, consider **urgent coronary angiography** when feasible and, if required, **percutaneous coronary intervention**.
 - o **Mechanical chest compression** devices and **extracorporeal CPR** can help facilitate this.
- **Pulmonary Embolism:**
 - o If PE is thought to be the cause of cardiac arrest consider giving a **fibrinolytic drug** immediately.
 - o Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR.
 - o If a fibrinolytic drug is given in these circumstances, consider performing CPR for **at least 60–90 min** before termination of resuscitation attempts.
 - o In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

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- **Tension pneumothorax:**

- Can be the primary cause of PEA and may be associated with trauma.
- The diagnosis is made clinically or by ultrasound.
- **Decompress rapidly** by thoracostomy or needle thoracocentesis, and then insert a chest drain.

- **Cardiac tamponade:**

- Usually difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself.
- Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for **resuscitative thoracotomy**.
- The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable.

- **Toxins:**

- In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations.
- Where available, the **appropriate antidotes** should be used, but most often treatment is supportive and standard ALS protocols should be followed.

D. USE OF ULTRASOUND IMAGING DURING ADVANCED LIFE SUPPORT

- Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death although sensitivity and specificity has not been reported.

E. WAVEFORM CAPNOGRAPHY DURING ADVANCED LIFE SUPPORT

- **Advantages:**

- **Ensuring tracheal tube placement in the trachea:** although it will not distinguish between bronchial and tracheal placement.
- **Monitoring ventilation rate during CPR:** avoiding hyperventilation.
- **Monitoring the quality of chest compressions** during CPR.
- **Identifying ROSC during CPR:** An increase in end-tidal CO_2 during CPR can indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC.
- **Prognostication during CPR:** The Resuscitation Council (UK) recommends that a specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

F. DEFIBRILLATION (MANUAL DEFIBRILLATORS)

- Continue chest compressions during defibrillator charging,
- Interruption in chest compressions of no more than 5 seconds
- Immediately resume chest compressions following defibrillation.
- Deliver the first shock with an energy of **at least 150 J**.
- If an initial shock has been unsuccessful it is worth attempting the second and subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy but, based on current evidence, both fixed and escalating strategies are acceptable.
- If VF/pVT recurs during a cardiac arrest (refibrillation) give subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy.

G. AIRWAY MANAGEMENT AND VENTILATION

- The options for airway management and ventilation during CPR vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers.
- They include:
 - No airway and No ventilation (compression-only CPR),
 - Compression-only CPR with the airway held open (with or without oxygen),
 - Mouth-To-Mouth breaths, Mouth-To-Mask, Bag-Mask Ventilation with simple airway adjuncts,
 - Supraglottic Airways (SGAs),
 - Tracheal Intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a SGA).
- Anyone attempting tracheal intubation must be well trained and equipped with waveform capnography.
- In the absence of these, use **bag-mask ventilation** and/or an **SGA** until appropriately experience and equipped personnel are present.

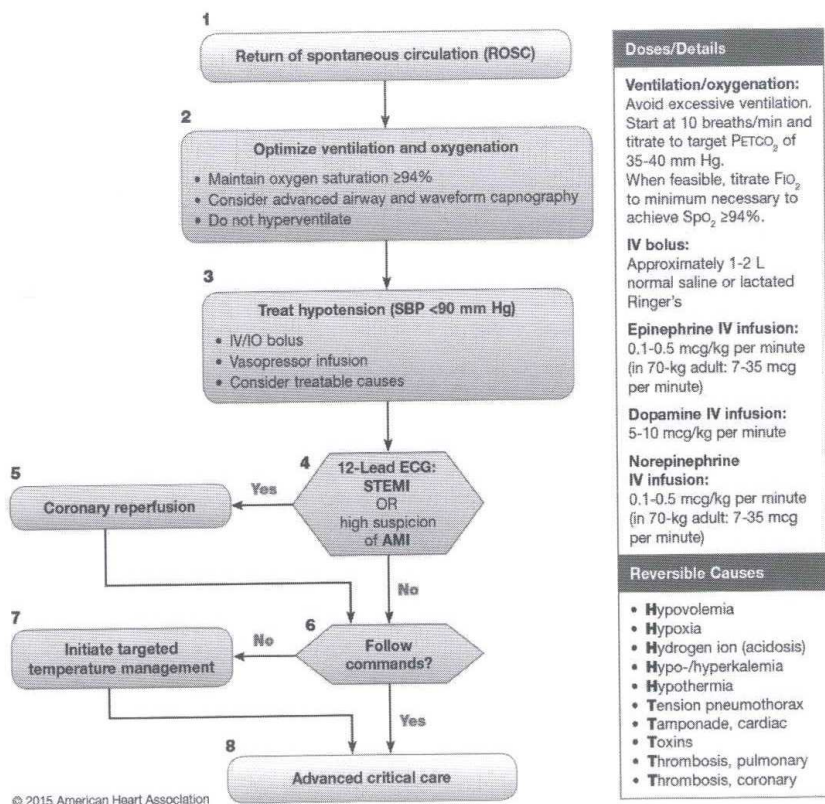
I. DRUGS FOR CARDIAC ARREST

- Although drugs are still included among ALS interventions, they are of secondary importance to high quality uninterrupted chest compressions and early defibrillation.*
- Vasopressin has been removed** from the algorithm altogether, and an emphasis on **EARLY** administration of epinephrine is stressed.*
- The **routine use of lidocaine after ROSC is not recommended**, however the initiation or continuation of lidocaine may be considered immediately after ROSC from ventricular fibrillation or pulseless ventricular tachycardia.*

1. ADRENALINE	2. AMIODARONE
<ul style="list-style-type: none"> Adult: Adrenaline 1 mg IV/IO Paeds: Adrenaline 10mcg/kg IV/IO Give as soon as possible in PEA/asystole. Give after the 3rd shock in VF/ VT. Repeat every 3–5 min (alternate cycles). 	<ul style="list-style-type: none"> Adult: Amiodarone dose 300 mg IV/IO bolus after the 3rd shock in VF/VT. Paeds: Amiodarone 5mg/kg A further 150 mg may be given for refractory VF/VT (5th shock) Followed by a 900mg infusion over 24 h.
<ul style="list-style-type: none"> If Amiodarone not available, give Lidocaine 1mg/kg IV 	
PAEDS ANTI-ARYTHMICS	
<ul style="list-style-type: none"> Amiodarone 5mg/kg –in paediatrics Lignocaine 1mg/kg – paediatrics Magnesium 0.1-0.2mmol/kg – paediatrics Atropine 1-3mg or 20mcg/kg – removed from adult PEA/asystole guidelines, still paediatrics NaHCO₃ 1mmol/kg – paediatrics 	

B. POST CARDIAC ARREST: CARE OF THE ROSC PATIENT

Adult Immediate Post-Cardiac Arrest Care Algorithm—2015 Update



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Figure 1.2.2. Post cardiac arrest care algorithm

- O₂ sats of 94–98% and normocapnia should be aimed for.
- A MAP of 65–100 should be aimed for.
- **Absent pupillary or corneal reflexes, absent or extensor motor responses to pain** are the best predictors of poor prognosis, but only at 72 hours after cardiac arrest.
- **Myoclonus status epilepticus** in the first day after cardiac arrest predicts poor prognosis.
- **Retention of any neurological function** (e.g. respiratory effort, coughing/swallowing, pupillary light reflex) after cardiac arrest is predictive of a good functional outcome.
- **An arterial lactate must be more than 16** to be 100% specific for poor neurological outcome.

C. RESUSCITATION IN SPECIAL CIRCUMSTANCES

1. OPIOID OVERDOSE

- In known opioid overdose associated with respiratory depression, respiratory arrest, or to help diagnose suspected opioid overdose, the usual initial adult dosage of **Naloxone Hydrochloride is 400–2000 mcg IV, given at 2–3 min intervals and titrated to response.**
- Naloxone may be given for cardiac arrest associated with opioid overdose, but its benefit is uncertain. **If no response is observed after a total of 10 mg IV Naloxone,** consider a non-opioid related drug or other process.
- If the IV route is not available, naloxone may be given by IM, IO, SC or intranasal routes.
- Additional doses may be necessary if the patient's level of consciousness falls, or if the patient's respiratory rate decreases again, because the half-life of naloxone can be shorter than the opioid causing the respiratory depression.
- Only give as much as is necessary to achieve an adequate respiratory rate, as an excessive dose, particularly in chronic opioid users, can cause **agitation and occasionally seizures.**

2. CARDIAC ARREST IN PREGNANCY

- **Causes of cardiac arrest in pregnancy:**
 - Haemorrhage,
 - Embolism (thromboembolic and amniotic fluid),
 - Hypertensive disorders of pregnancy,
 - Abortion
 - Genital tract sepsis
- **Differential diagnosis for chest pain/cardiac arrest in pregnancy:**
 - Pulmonary embolism
 - Aortic dissection
 - ACS
 - Spontaneous Coronary Artery Dissection (**SCAD**) (21% of AMI post partum)
 - Arrhythmia including Long QTc
- **Approach of cardiac arrest in pregnancy**
 - Use the **ABCDE approach** and follow ALS algorithm
 - **Identify and treat the underlying cause** (e.g. rapid recognition and treatment of sepsis, including early intravenous antibiotics).
 - Place the patient in the **left lateral position** or **manually displace the uterus to the left.**
 - Give **high-flow oxygen**, guided by pulse oximetry and aim to correct hypoxaemia.
 - Establish **IV access and give a fluid bolus** (250 mL) if there is hypotension or hypovolaemia.
 - Seek **expert help early:** Obstetric, anaesthetic and neonatal specialists should be involved early in the resuscitation.
 - **Defibrillation** energy levels are as recommended for standard defibrillation. If left lateral tilt and large breasts make it difficult to place an apical defibrillator electrode, use an **antero-posterior or bi-axillary electrode position.**
 - If resuscitation attempts fail to achieve ROSC, consider an **immediate caesarean section to deliver the fetus.**

3. TRAUMATIC CARDIAC ARREST**• REVERSIBLE CAUSES OF TRAUMATIC CARDIAC ARREST**

- Hypovolaemia,
- Hypoxia (Oxygenation)
- Tension pneumothorax
- Tamponade – cardiac

- Patients with traumatic cardiac arrest commonly have one or more injuries resulting in severe hypovolaemia, critical hypoxaemia, tamponade or tension pneumothorax, either in isolation or concurrently.
- Each of these conditions needs to be addressed simultaneously by the prehospital team and active management commenced.

• HYPOVOLAEMIA AND FLUID REPLACEMENT

- Immediately control active external haemorrhage by applying **direct pressure** to bleeding wounds.
- Then **volume re-expansion** should follow.
- **Splint fractures** of the pelvis and long bones and if there is a suspicion of a pelvic fracture, apply a **pelvic binder** to reduce the pelvis to an anatomical position taking care to minimise patient movement.
- **Reduce long bone fractures** to an anatomical position and apply splints.
- **Tranexamic Acid**
 - Give adult trauma patients with suspected haemorrhage a prehospital dose of **Tranexamic acid 1g IV/IO over 10 min.**

• HYPOXAEMIA

- Initial attention should be paid to high quality, basic airway management **with cervical spine control**, using airway adjuncts if required.
- Attention to basic airway management is vital in the unconscious trauma patient who is at risk of airway compromise.
- Secure a definitive airway by insertion of a **cuffed tracheal tube** as early as possible.

• TENSION PNEUMOTHORAX

- Manage any open pneumothorax or sucking chest with a **dressing** that enables air to be released from the pleural cavity.
- **Bilateral needle chest decompression** is rapid and within the skill set of most EMS personnel and should be performed immediately.
- Tracheal intubation, positive pressure ventilation and formal chest decompression will effectively treat tension pneumothorax in patients with traumatic cardiac arrest.
- **Simple thoracostomy** is straightforward and used in several prehospital physician services.

4. ASTHMA

- If IV or IO access cannot be established rapidly, give **IM adrenaline** if cardiorespiratory arrest has occurred recently. When the appropriate skills are available **intubate** the trachea to enable ventilation of stiff lungs and avoid gastric insufflation.
- **Identify and treat tension pneumothorax** with needle decompression or thoracostomy as appropriate. Cardiac arrest associated with asthma results from respiratory exhaustion, respiratory acidosis and impaired venous return caused by high intrathoracic pressures.

- It may also be precipitated by a tension pneumothorax that is, on rare occasions, bilateral.
- If there is a history of a severe asthma attack leading to cardiac arrest, **adrenaline 0.5 mg IM can be given early**, if IV access is not immediately available.

5. HYPOXIA

- Cardiac arrest caused by pure hypoxaemia is uncommon.
- It is seen more commonly as a consequence of asphyxia, which accounts for most of the non-cardiac causes of cardiac arrest.
- **Causes of asphyxial cardiac arrest:**
 - Airway obstruction: soft tissues (coma), laryngospasm, aspiration
 - Anaemia
 - Asthma Avalanche burial
 - Central hypoventilation – brain or spinal cord injury Chronic obstructive pulmonary disease
 - Drowning
 - Hanging High altitude
 - Impaired alveolar ventilation from neuromuscular disease Pneumonia
 - Tension pneumothorax
 - Trauma
 - Traumatic asphyxia or compression asphyxia (e.g. crowd crush)
- **Treatment**
 - Effective ventilation with supplementary oxygen

6. HYPERKALAEMIA

- Hyperkalaemia is the most common electrolyte disorder associated with life threatening arrhythmias and cardiac arrest. It is defined as $K_{(s)} > 5.0 \text{ mmol/l}$.

Mild	5.0-5.9 mmol/l
Moderate	6.0-6.4 mmol/l
Severe	> 6.5 mmol/l

- Mild hyperkalaemia is common and often well tolerated in patients with chronic renal failure. $K_{(s)} > 10 \text{ mmol/l}$ is usually fatal.
- **CLASSIC CAUSES OF HYPERKALAEMIA**
 - **Drugs**
 - Angiotensin converting enzyme inhibitors (ACEI)
 - Angiotensin receptor blockers (ARB)
 - Non-steroidal anti inflammatories (NSAIDs)
 - Beta blockers
 - Suxamethonium
 - K^+ supplementation
 - K^+ sparing diuretics
 - **Renal and metabolic diseases**
 - Acute and Chronic Renal Failure
 - Type 4 Renal Tubular Acidosis

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- Metabolic acidosis
- Diet
- Fasting caused by a relative lack of insulin
- **Endocrine disorders**
 - Addison's disease
 - Hypoparathyroidism
 - Insulin deficiency
- **Others**
 - Tumour lysis
 - Rhabdomyolysis
 - Massive transfusion
 - Massive haemolysis
 - Haemolysis (in laboratory tube)
 - Thrombocytosis
 - Leukocytosis
 - Venepuncture technique (e.g. prolonged tourniquet application)
- **CLINICAL MANIFESTATIONS**
 - Patients with hyperkalaemia frequently appear well.
 - The following symptoms usually occur in severe cases but are very non-specific:
 - Flaccid paralysis
 - Paraesthesia
 - Respiratory difficulties
 - Signs such as depressed deep tendon reflexes
 - Arrhythmias: VT, VF, PEA...
- **Bradycardia** is also common in hyperkalaemia and causes a dilemma in that calcium salt administration can worsen the situation. The response to atropine is also poor.

ECG IN HYPERKALAEMIA

- **Serum potassium > 5.5 mEq/L** is associated with **repolarization abnormalities**:
 - Peaked T waves (usually the earliest sign of hyperkalaemia)
- **Serum potassium > 6.5 mEq/L** is associated with **progressive paralysis of the atria**:
 - P wave widens and flattens
 - PR segment lengthens
 - P waves eventually disappear
- **Serum potassium > 7.0 mEq/L** is associated with **conduction abnormalities and bradycardia**:
 - Prolonged QRS interval with bizarre QRS morphology
 - High-grade AV block with slow junctional and ventricular escape rhythms
 - Any kind of conduction block (bundle branch blocks, fascicular blocks)
 - Sinus bradycardia or slow AF
 - Development of a sine wave appearance (a pre-terminal rhythm)
- **Serum potassium level of > 9.0 mEq/L** causes **cardiac arrest** due to:
 - Asystole
 - Ventricular fibrillation
 - PEA with bizarre, wide complex rhythm

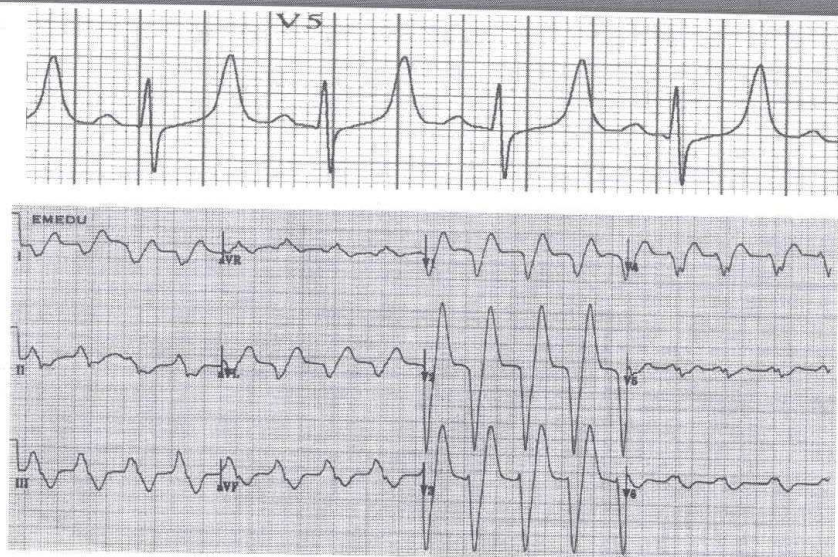


Figure 1.2.3. ECG in Hyperkalaemia

EM MANAGEMENT OF HYPERKALAEMIA

- o Treatment of hyperkalaemia involves stabilizing the myocardium to prevent arrhythmias, shifting potassium back into the intracellular space and removing excess potassium from the body.

Mechanism	Drug/ Method	Dose	Onset (min)	Duration (hr)
Stabilizing membranes	Calcium chloride	10ml 10% IV	1-30	0.5-1
Shift K	Insulin/Glucose	10U in 100ml of dextrose 10%	15-30	4-6
	Salbutamol	0.5mg IV 20mg Nebs	15-30	4-6
	Na Bicarbonate	1mmol/kg IV	15-30	Several
Remove excess K	Calcium resonium	15-30g PO/PR	Variable	4-6
	Dialysis	Most immediate and reliable method of K removal Can lower potassium by 1mmol/L in first hour and another 1mmol/L over the next 2 hours.		

INDICATIONS FOR DIALYSIS.

- o The main indications for dialysis in patients with hyperkalaemia are:
 - Severe life-threatening hyperkalaemia with or without ECG changes or arrhythmia;
 - Hyperkalaemia resistant to medical treatment;
 - End-stage renal disease;
 - Oliguric acute kidney injury (<400 mL/day urine output);
 - Marked tissue breakdown (e.g. rhabdomyolysis).

7. HYPOKALAEMIA

- Hypokalaemia is defined as $K_{(s)} < 3.5 \text{ mmol/l}$, symptoms are more likely with increasing severity.

Mild	3.0-3.5 mmol/l
Moderate	2.5-3.0 mmol/l
Severe	< 2.0 mmol/l

- Causes:**
 - The most common cause of hypokalaemia is **potassium depletion**.
 - In critically ill patients the most common cause is **abnormal losses** which occur in stool and urine (from metabolic alkalosis and chloride depletion).
 - Other causes of hypokalaemia are:**
 - Gastrointestinal loss** (e.g. Diarrhoea, vomiting, ileostomy, intestinal fistula);
 - Drugs** (e.g. Diuretics, laxatives, steroids);
 - Renal losses** (e.g. Renal tubular disorders, diabetes insipidus, dialysis);
 - Endocrine disorders** (e.g. Cushing's/conn's syndromes, hyperaldosteronism);
 - Transcellular shift:** Insulin/Glucose, Theophylline, Caffeine, Hyperthyroidism
 - Metabolic alkalosis;** Magnesium depletion; Poor dietary intake.

ED MANAGEMENT OF HYPOKALAEMIA**1. MILD/MODERATE HYPOKALAEMIA**

- Dietary supplementation and monitoring may suffice.
- Gradual Potassium administration.
- Magnesium supplementation facilitates more rapid correction of hypokalaemia.

2. SEVERE HYPOKALAEMIA

- In severe hypokalaemia, intravenous replacement must be used.
- This must be rigorously controlled using infusion pumps according to local protocols.
- The maximal rate of correction is **20 mmol/h K^+** .
- Magnesium 5 ml of 50% over 30 minutes** should commence soon after.
- Never bolus inject potassium and always ensure adequate mixing of the solution occurs before the infusion is started.

3. CARDIAC ARREST

- Cardiac arrest due to hypokalaemia may require **20 mmol potassium chloride IV over 2-3 minutes, repeated until potassium is > 4.0 mmol/l**.
- Prompt correction increases the chances of successful defibrillation and may decrease the incidence of post arrest arrhythmias.

ECG features of hypokalaemia are:

- U waves** Prominent;
- T wave** flattening;
- ST segment** depression
- PR interval** prolonged
- P wave** slightly peaked

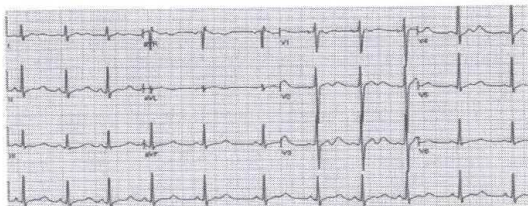


Figure 1.2.4. ECG in Hypokalaemia

8. HYPOTHERMIA

INTRODUCTION

- Hypothermia is the pathological state in which the core body temperature falls $< 35^{\circ}\text{C}$.
- Hypothermia can be further subdivided into **Mild ($35\text{--}32^{\circ}\text{C}$)**, **Moderate ($32\text{--}30^{\circ}\text{C}$)**, **Severe ($30\text{--}20^{\circ}\text{C}$)** and **Profound ($<20^{\circ}\text{C}$)**.
- Hypothermia is classified as either primary or secondary:
 - **Primary hypothermia** occurs when an otherwise healthy individual is exposed to environmental circumstances, such as adverse weather or cold water immersion that causes his/her core temperature to drop.
 - **Secondary hypothermia** occurs when the drop-in core temperature is secondary to a primary disease process such as alcohol intoxication, trauma or a myocardial infarct.

Aetiology/causes

General

- Young and old
- Systemic illness
- Sepsis
- Malnutrition

Environmental

- Cold, wet, windy conditions
- Cold water immersion
- Exhaustion
- Marathon runners

Trauma

- Multiple trauma
- Minor trauma, immobility (e.g. # NOF)
- Major burns

Drugs

- Ethanol
- Sedatives (e.g. BDZ, TCAs, opioids OD)
- Phenothiazines (impaired shivering)

Neurological

- CVA
- Paraplegia
- Parkinson's disease

Endocrine

- Hypoglycaemia and diabetes
- Hypothyroidism
- Hypoadrenalism

CLINICAL ASSESSMENT:

- In determining whether hypothermia is playing a significant role in your patient's presentation consider:
 - Where they were found
 - The ambient temperature and weather conditions
 - The patient's clothing
 - The patient's age
 - Co-morbid conditions and state of nutrition
 - Alcohol and drug use
- **Sinus bradycardia** develops followed by atrial fibrillation (AF).
- **Below 32°C** , ventricular arrhythmias including ventricular fibrillation (VF) may occur. Finally, asystole results.
- Note that malignant arrhythmias are unlikely to be hypothermia-induced at temperatures above 32°C – consider alternative causes such as acute coronary syndrome (ACS).
- The rhythm strips below show the different stages of hypothermia

- **DIFFERENTIAL DIAGNOSIS:**

- o All that is needed to make a diagnosis of hypothermia is a low temperature recorded on an accurate thermometer.
- o The diagnosis can be easily missed by not obtaining a full set of vital signs on the patient or by being misled by an inaccurate tympanic or oral thermometer.
- o If hypothermia is suspected, a **rectal temperature** should be performed to confirm the diagnosis.
- o The diagnosis of hypothermia may also be suspected by **Osborn or J waves on ECG**.
- o The upward deflection of the terminal S wave (at the junction of the QRS and the ST segment) occurs at or near 32°C. It is **first seen in leads II and V6**.

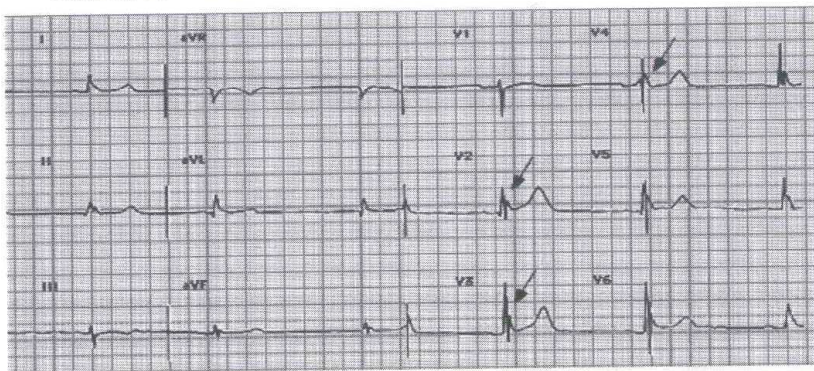


Figure 1.2.5. Osborn or J waves on ECG.

MANAGEMENT OF HYPOTHERMIA IN THE ED

- **General Approach**

- o **ABC approach** including **Dont Ever Forget Glucose**
- o Removal of wet, cold clothes is the cornerstone of management
- o Prevention of further heat loss;
- o Initiation of re-warming appropriate to the degree of hypothermia
 - The patient must be placed on a cardiac monitor,
 - Intravenous access established
 - Active re-warming measures initiated.
- o Treatment of complications and other medical factors (such as alcohol intoxication, central nervous system disease, trauma and infection should be considered and treated concurrently).
- o *The general treatment of any coagulopathies is by re-warming and not the administration of clotting factors.*

- **Additional Diagnostic Measures:** ABG, FBC, U&E, Clotting screen, CK, Blood alcohol and a urine toxicology screen.

- **Some of the physiological changes seen with hypothermia would include:**

- o Shift of the oxyhaemoglobin dissociation curve to the left,
- o Increased haematocrit due to the decrease in circulating plasma volume
- o Low to normal white blood cell count, even in the setting of infection.
- Dont correct for hypothermia when analysing the acid-base status, normal values can be assumed to meet the needs of the hypothermic tissue.

- **Temperature Monitoring**
 - o This should be accomplished by the use of a continuous or serial rectal or oesophageal thermometer.
- **Fluid Resuscitation**
 - o In general, the hypothermic patient is dehydrated and fluid depleted.
 - o They should therefore be given a fluid challenge of **warmed 0.9% saline** or **preferably Dextrose-Saline** as they may also be hypoglycaemic.
 - o *Hartmanns solution may also be used but since the hypothermic liver cannot metabolise lactate, it is best avoided.*
 - o The patient should be monitored carefully for signs of fluid overload.

1. PASSIVE REWARMING

- o Remove patient from the cold environment
- o Wet or cold clothes removed.
- o Cover with a blanket or sleeping bag
- o Covered their head to reduce heat loss.

2. ACTIVE REWARMING

A. ACTIVE EXTERNAL REWARMING:

- Treatment of choice in **mild-moderate hypothermia**
- Heat packs, heat lamps, and blankets, warm water immersion, warmed blankets, and forced air systems.

B. ACTIVE CORE REWARMING:

- Patients with **moderate to severe hypothermia** will require active core rewarming
- Use of **warmed IV Fluids at 44°C.**
- **Warmed humidified air/oxygen heated to 42-44°C.**
- Gastric, Bladder, Peritoneal and Pleural lavage using warm fluids

C. EXTRACORPOREAL BLOOD REWARMING:

- Haemodialysis, Arteriovenous, Veno-venous and Cardio-pulmonary bypass.

- o There are no specific criteria for placing a patient on extracorporeal rewarming, but several centres reserve it for patients with a **pH >6.5**, a **serum potassium <10mmol/L**, and a **core temperature > 12°C.**

• DISPOSITION

- o **Mild hypothermia** who respond to passive rewarming are usually able to be discharged from the Emergency Department (ED).
- o **Severe hypothermia** need to be admitted to a HDU/ITU setting.
- o Patients with **Moderate hypothermia** are the most difficult. Their disposition will depend on the patient's age, comorbid factors, social situation and response to ED treatment.

HYPOTHERMIC CARDIAC ARREST

• Defibrillation and Pacing

- **Defibrillation is less effective in hypothermia.** For ventricular fibrillation/ventricular tachycardia (VF/VT) defibrillation may be tried up to three times but is then not tried until the temperature reaches 30°C.
- **Pacing is generally ineffective.** Do not try it unless bradycardia persists when normothermia is reached. Sinus bradycardia may be a physiological response and is not treated specifically.

• Ventilation

- Normocapnia will be achieved at lower minute volumes than normal and hyperventilation risks cerebral hypoxia through reduction of cerebral blood flow.
- Aim for a normal CO₂ on ABG (**not** corrected for the patient's temperature).

• Intubation

- In a patient with a perfusing rhythm, intubation (or other rough handling of the patient) may precipitate VF, although the evidence for this is mainly animal-based and it is rare.

• Resuscitation drugs

- Drugs are often ineffective and will undergo reduced metabolism; so these are **withheld below 30°C then given with twice the time interval between doses** until either normothermia is approached or circulation restored.
- *So, adrenaline would be given about every 8-10 minutes once the core temperature is above 30°C.*

• Chest compressions

- Hypothermia causes muscular stiffness: chest compressions may be harder work than normal.
- Make sure that the individual performing chest compressions is swapped frequently.

Conclusion

- Patients with a **Potassium level over 10 mmol/L** or **severe traumatic injuries** will not benefit from bypass.
- Similarly, patients with **pre-existing cardiopulmonary, renal or neurological disorders** require careful selection as they have a poorer prognosis.
- In primary hypothermic cardiac arrest, **death should not be confirmed until:**
 - *The patient has been re-warmed or*
 - *Other unsurvivable injuries have been identified or*
 - *Re-warming has failed despite all available measures*

"Nobody is dead until warm and dead"

CHAPTER 3. SEPTIC PATIENT

1. ADULT SEPSIS SIX PATHWAYS

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)

- **SIRS criteria is met if 2 or more are present:**
 - **Temperature** > 38°C or < 36°C.
 - **Pulse** > 90 beats/min.
 - **Respiratory Rate (RR)** > 20 or $\text{PaCO}_2 < 4.3 \text{ kPa}$.
 - **WBC** > 12,000 or < 3000/mm³ (or > 10% immature bands).
 - **Acutely Altered Mentation.**
 - **Blood glucose** > 6.6 (no Hx diabetes).
- **Lactate in Severe Sepsis**
 - Hi Lactate (& rate of clearance) is prognostic
 - **Initial Lactate:**
 - **0-2 Normal.**
 - **>2 (If criteria for sepsis) = Severe Sepsis.**
 - **>4 (If criteria for sepsis) = Septic shock.**
 - **>4 Septic Shock** (NB if BP was never low then = '**Cryptic Shock**'). If, despite initial resuscitation (O₂, fluids, swabs & cultures, antibiotics, blood tests and urinary catheter for hourly U/O), the BP remains low (SBP<90, MAP<65) then this is **Septic Shock irrespective of the Lactate.**

DEFINITIONS

- **Sepsis** is "the systemic inflammatory response syndrome (SIRS) during an infection."
- **Severe sepsis:** Sepsis and at least 1 organ dysfunction:
 - **Skin:** Areas of mottled skin or Cap Refill Test >3sec.
 - **Neurological:** New altered mental status.
 - **Haematologic:** Platelets < 100,000; INR >1.5; PTT >60 sec
 - **Renal:** creatinine > 2.0 mg/dL without prior chronic renal disease; or increase 0.5 mg/dL; acute oliguria urine output <0.5 mL/kg/hr for at least 2 hours despite fluid resuscitation.
 - **Pulmonary:** RR > 20, oxygen (O₂) saturation < 90% or < 94% with supplement O₂, or mechanical ventilation.
 - **GI:** Ileus; absent bowel sounds; hyperbilirubinaemia plasma total bilirubin >4 mg/dL.
 - **Cardiovascular:** Shock.
- **SEPTIC SHOCK**
 - Sepsis and refractory hypotension defined as systolic blood pressure < 90 mm Hg, mean arterial pressure (MAP) < 65 mm Hg, or decrease of 40 mm Hg in systolic pressure compared with baseline; **unresponsive to crystalloid fluid challenge of 20 to 40 mL/kg.**
- **BACTERAEMIA**
 - Presence of viable bacteria in the blood; found in about 50% of cases of severe sepsis and septic shock; whereas 20% to 30% of patients will have no cause identified from any source.

- **NEUTROPENIA**

- An abnormal decrease in the number of neutrophils in the blood.
- **Neutropenic sepsis** is diagnosed in patients having anti-cancer treatment who present unwell with a **neutrophil count 0.5×10^9 or lower, or less than 1×10^9 with a downward trend.**
- Neutropenia is a common problem in oncology patients either following chemotherapy, or less commonly secondary to radiation treatment or marrow infiltration by malignancy.
- Neutropenia is most likely to occur **10-14 days post-chemotherapy** but should remain a consideration after this period.

- **FEBRILE NEUTROPENIA**

- Occurs when a patient has a fever and a significant reduction in their neutrophil counts.
- The fever may be caused by an infectious agent, and when it is, prompt treatment is required.
- A patient with febrile neutropenia needs assessment for the possible source, type of infection and treatment until the cause is found or it subsides.

ED MANAGEMENT OF SEPSIS

- **Initial Resuscitation**

SEPSIS SIX BUNDLE	
TAKE 3	GIVE 3
<ol style="list-style-type: none"> 1. CULTURES: Take blood cultures before giving antimicrobials (if no significant delay i.e. >45 minutes) and consider source control. 2. BLOODS: Check lactate and FBC (Full Blood Count). 3. URINE OUTPUT: Assess urine output and consider urinary catheterisation for accurate measurement in patients with severe sepsis/septic shock. 	<ol style="list-style-type: none"> 1. OXYGEN: Titrate O₂ supplementation to saturations of 94 -98% or 88-92% in chronic lung disease. 2. FLUIDS: Start IV fluid resuscitation if evidence of hypovolaemia and/or shock. 500ml-1000mls bolus of isotonic crystalloid over 15-30 minutes and give up to 30ml/ kg, reassessing after each bolus for signs of hypovolaemia, euvolaemia, or fluid overload. 3. ANTIMICROBIALS: Give IV antimicrobials according to local antimicrobial guidelines

Recommendation (3 Hour Bundle)

- For Patients with Severe Sepsis/Septic Shock to be Completed **Within 3 Hours** Of Diagnosis:
 - 1. Complete Sepsis 6 within first hour.**
 - 2. Administer a minimum of 30 mL/kg isotonic crystalloid for hypotension or lactate >4mmol/L**
 - 3. Assess patient for response to resuscitation by monitoring clinical and haemo-dynamic response, measure hourly urinary output and repeat lactate measurement.**
- **Resuscitation goals:**
 - Central venous pressure: 8-12 mm Hg.
 - Mean arterial pressure ≥ 65 mm Hg.
 - Urine output ≥ 0.5 mL / kg / hr.
 - Central venous or mixed venous oxygen saturation $\geq 70\%$.

- If central venous oxygen sat. or mixed venous O_2 sat. of 70% is not achieved (with a CVP 8-12 mm Hg), then transfuse packed red blood cells to haematocrit $\geq 30\%$ **and/or** administer a **Dobutamine infusion** (up to max of $20 \mu\text{g} / \text{kg} / \text{min}$).
- **Source control**
 - Look for the source of infection: ? Abscess drainage / tissue debridement.
 - Choose the source control measure that will cause the least physiological upset and still accomplish the clinical goal.
- **Vasopressors**
 - **Start vasopressors when fluid challenge fails to restore adequate blood pressure and organ perfusion.**
 - **Norepinephrine or Dopamine** (via central line) are the vasopressors of choice.
 - Consider small boluses (ask your senior first) of 1:100,000 Adrenaline while setting up the NA infusion. Titrate vasopressors to MAP of $>65 \text{ mmHg}$.
 - Do **not** use low-dose Dopamine for renal protection
 - Place **an arterial line**. Vasopressin can be considered later (after transfer to ITU).
- **Steroids**
 - Treat patients who still require vasopressors despite fluid replacement with hydrocortisone (200-300 mg/day) **Or**
 - Perform 250-microgram ACTH Stimulation Test and discontinue steroids in responders.
- **Fluid therapy**
 - Give 500-1000 ml of **crystalloid** over 30 mins.
 - Repeat if BP and urine output do not increase (with no evidence of intravascular volume overload).
- **Blood products**
 - Once tissue hypoperfusion improved (and no significant coronary artery disease or acute haemorrhage), transfuse with red blood cells to a target a haemoglobin of 7.0 - 9.0 g/dL.
 - Do **not** use (FFP) Fresh Frozen Plasma to correct laboratory clotting abnormalities, unless there is bleeding or planned invasive procedures.
 - Do **not** use antithrombin therapy.
 - Administer platelets when counts are less than $5000/\text{mm}^3$ ($5 \times 10^9/\text{L}$), regardless of bleeding.
 - Transfuse platelets when counts are 5000 to $30,000/\text{mm}^3$ ($5-30 \times 10^9/\text{L}$) and there is significant bleeding risk.
 - Higher platelet counts ($= 50,000/\text{mm}^3$ [$50 \times 10^9/\text{L}$]) are required for surgery or invasive procedures.
- **Glucose control**
 - Maintain BM $< 8.3 \text{ mmol/L}$ following initial stabilization - insulin +/- glucose) infusion.
- **Renal replacement**
 - Do **not** use **bicarbonate** therapy to improve haemodynamics (e.g. "lactic acidosis")
- **DVT**
 - Use either low-dose unfractionated heparin or LMWH.
- **Provide stress ulcer prophylaxis.**
 - With H_2 receptor inhibitors.

2. PAEDIATRIC SEPSIS SIX PATHWAYS
**THE UK
SEPSIS
TRUST**
Paediatric Sepsis 6

Date of Birth:

Hospital No.:

*Affix Hospital Label if available***Recognition of a child at risk:****If a child with suspected or proven infection AND has at least 2 of the following:**

- Core temperature < 36°C or > 38.5°C
- Inappropriate tachycardia (Refer to local criteria / APLS Guidance)
- Altered mental state (including: sleepiness / irritability / lethargy / floppiness)
- Reduced peripheral perfusion / prolonged capillary refill

Lower threshold of suspicion for: age < 3 months, chronic disease, recent surgery, or immunocompromised

THINK: Could this child have SEVERE SEPSIS, SEPTIC SHOCK or RED FLAG SEPSIS* - Ask for review by an experienced clinician.

 High certainty of Sepsis
Respond with Paediatric Sepsis 6:

 High certainty NOT Sepsis
or Unsure
Complete all elements within 1 hour**1. Give high flow oxygen:****2. Obtain IV / IO access & take blood tests:**

- Blood cultures
- Blood glucose - treat low blood glucose
- Blood gas (+ FBC, lactate / CRP as able)

3. Give IV or IO antibiotics:

- Broad spectrum cover as per local policy

4. Consider fluid resuscitation:

- Aim to restore normal circulating volume and physiological parameters
- Titrate 20 ml/kg Isotonic Fluid over 5 - 10 min and repeat if necessary
- Caution with fluid overload:
Examine for crepitations & hepatomegaly

5. Involve senior clinicians / specialists early:**6. Consider inotropic support early:**

- If normal physiological parameters are not restored after ≥ 40 ml/kg fluids
- NB adrenaline or dopamine may be given via peripheral IV or IO access

Document reason(s) for variation overleaf

 Not Sepsis
Document reasons
Unsure
Review within 1 hour
 Not Sepsis
Document reasons
Sepsis
Start Sepsis 6
Unsure
Review within 1 hour
 Not Sepsis
Document reasons
Sepsis
Start Sepsis 6
Unsure
Review within 1 hour
 Not Sepsis
Document reasons
Sepsis
Start Sepsis 6
Unsure
Review within 1 hour
 Not Sepsis
Document reasons
Sepsis
Start Sepsis 6

CHAPTER 4. SHOCKED PATIENT

• DEFINITION

- **Shock** is a life-threatening condition that results when circulatory insufficiency leads to inadequate tissue perfusion and thus delivery of oxygen to the tissues of the body.
- Shock can be the result of numerous different pathophysiological processes that can be broadly accommodated within 4, somewhat artificial categories:
 - **Hypovolaemic:** Haemorrhage, Diarrhoea and Vomiting, DKA, Burns
 - **Distributive:** Septic, Anaphylactic and Neurogenic shocks
 - **Obstructive:** Tension Pneumothorax, PE, Cardiac tamponade and IVC/SVC obstruction
 - **Cardiogenic:** Myocardial infarction/contusion, Myocarditis, Late sepsis, complete heart block, OD β -Blockers

BASIC SCIENCE AND PATHOPHYSIOLOGY

• What determines oxygen delivery (DO_2)?

- Global oxygen delivery is determined by **Cardiac Output (CO)** and **Arterial Oxygen content (CaO_2)**. $\text{CO} = \text{HR} \times \text{SV}$ (Heart rate x Stroke Volume).
- For practical purposes, global oxygen delivery can be calculated as:
 $\text{GOD} = (\text{HR} \times \text{SV}) \times [\text{Hb}] \text{g/dl} \times 10 \times 1.34 \times \text{sO}_2 \text{ ml/l}$
 - The 10 is to convert g/dl of Hb to g/l.
 - The 1.34 represents the amount of oxygen (in ml) carried by one gram of 100% saturated haemoglobin.

The top 3 interventions increasing oxygen delivery by almost 100% are:

- **Increasing Hb** is the (single most effective intervention)
- **Increasing cardiac output** (Blood transfusion would in reality achieve both).
- **Ensuring oxygen saturation** is maximal is somewhat effective while achieving supranormal PaO_2 achieves little.

• CLINICAL ASSESSMENT

- Assessment and management of the patient must follow an **ABCDE** approach and involve 4 key steps that should ideally occur concurrently:
 - Recognition of the degree of physiological compromise
 - Identification of the cause
 - Correction of the physiological deficit
 - Treatment of the underlying cause

• CLASSIFICATION OF SHOCK

Class of shock	Class I	Class II	Class III	Class IV
Volume Blood loss (ml)	Up to 750	750-1500	1500-2000	>2000
Volume of blood loss (%)	0-15%	15-30%	30-40%	>40%
Heart Rate	<100	>100	>120	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory Rate	14-20	20-30	30-40	>35
Mental State	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

- Note also a reduction in pulse pressure occurs before a reduction in systolic BP as the diastolic increases in response to vasoconstriction.

28 FRCEM Intermediate

- The mean arterial pressure ($MAP = (systolic + 2 \times diastolic) / 3$) is a better representation of organ perfusion than the systolic.
- A MAP of 65mmHg is considered to be sufficient for organ perfusion in a healthy adult.

• Other history and examination findings:

Finding	Possible cause
○ Pain radiating to testicle	○ Abdominal aortic or iliac aneurysm
○ Chest pain radiating to back	○ Thoracic aortic dissection
○ Onset with food	○ Anaphylaxis
○ History of active rheumatoid arthritis	○ Addisonian crisis
○ Muffled heart sounds	○ Cardiac tamponade
○ Priapism	○ Neurogenic shock
○ Unequal radial pulses	○ Thoracic aortic dissection
○ Distended neck veins	○ Tension pneumothorax
○ Sweet smelling breath	○ Diabetic ketoacidosis

• INVESTIGATION STRATEGIES

- The majority of the investigations in a shocked patient will be focused on identifying the cause of the shock (e.g. FAST scan in trauma, ECG in cardiogenic shock, echocardiography in massive pulmonary embolus).
- **ABG: lactate, Anion gap, Base excess...**
- **Central venous oxygen saturation:** Don't be falsely reassured by a normal ScvO₂ it may simply represent the tissues inability to utilise oxygen.

MANAGEMENT OF A SHOCKED PATIENT IN THE ED

- Once a shock state is recognised treatment must focus on:
 - *Reversing the physiological deficit (resuscitation)*
 - *Treating the cause.*
- Resuscitation and definitive treatment should be contemporaneous and must be tailored to the specific diagnosis.
- **General management:**
- **MOVER: Monitor, Oxygen, Vital Signs, ECG, Resus**
 - **A: Patent airway**
 - **B: Maximise oxygen delivery**
 - Consider early **intubation and ventilation** in many shocked patients.
 - **C: 2 large bore IV Cannula;**
 - Get blood: **ABG, FBC, U&E, LFT, CRP, Blood Cultures, Cross match, tryptase...**
 - **IV fluid** (crystalloids) bolus: Small volumes (eg 250ml) given quickly (over 5-10 min).
 - Consider **intubation** once fluid resuscitation **exceeds 40-60 ml/kg.**
 - **Judicious transfusion:** reasonable target: **7-9 g/dl** in otherwise healthy patients.
 - **D: Inotropes** have a role in some conditions (eg sepsis, cardiogenic shock, neurogenic shock, anaphylaxis) but are likely to be harmful in other settings.
 - Exactly which inotrope in which setting is a subject of vigorous ongoing debate.
 - With the exception of **adrenal insufficiency (Addisonian crisis)** which should be considered in all hypotensive patients where there is no apparent cause, particularly those on corticosteroids and if there is both unexplained hyponatraemia and hyperkalaemia, **there is no role for steroid use** in the initial resuscitation and treatment of a shocked patient.

CHAPTER 5. UNCONSCIOUS PATIENT

I. COMA

OVERVIEW

- A useful way of approaching the unconscious patient is to use these three categories:
 - Coma with focal or lateralising signs
 - Coma without focal or lateralising signs but with meningism
 - Coma without either focal or lateralising signs or meningism

CAUSES OF COMA

- Mnemonics "TIPS AEIOU"
 - Trauma to head
 - Insulin: too little or or too much
 - Psychogenic
 - Stroke
 - Acidosis/ Alcohol
 - Epilepsy
 - Infection
 - Overdose
 - Uremia

DIFFERENTIAL DIAGNOSIS:

Head Injury

- Maintain adequate CPP and Oxygenation
- Urgent CT Head and possible other regions

No Head Injury But Focal Neurology

- Probable cause: **CVA** (Requires urgent CTB)
- Exclude **Hypoglycaemia**

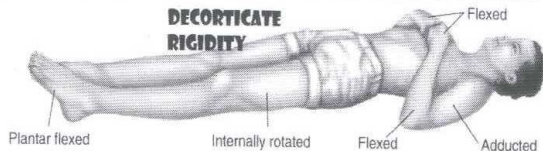
No Head Injury, No Focal Neurology But evidence of Neck Stiffness

- Probable cause: **Meningitis**
- Exclude **Cerebral Malaria**: History \pm specific tests
- Exclude **Cerebellar Haemorrhage**: clinic; \pm CT/MRI

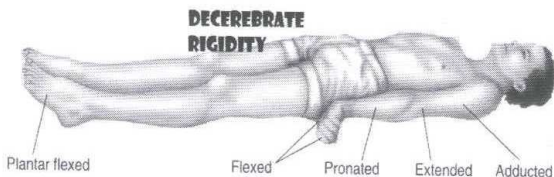
No head Injury, No Focal Neurology No Neck Stiffness

- Exogenous Poisons**: Carbon monoxide, Tricyclic antidepressants, Narcotics
- Endogenous Poisons**: Diabetic Ketacidosis, Myxoedema coma, Respiratory failure

DECORTICATE RIGIDITY



DECEREBRATE RIGIDITY



Both are poor prognostic signs

- Patients with arms flexed and legs extended are said to have a **decorticate posture**.
- This indicates **injury above the midbrain**.
- Those with legs and arms extended and adducted are said to have a **decerebrate posture**.
- This indicates a **brainstem lesion**.

- Following the above focused examination coma patients can be assigned to one of four groups which then guides further assessment, investigation and management
 - Evidence of Head Injury
 - No head injury but focal neurological signs
 - No head injury, no focal neurological signs, infection probable (History, Temp, WBC)
 - No head injury, no focal neurological signs, infection unlikely (History, Temp, WBC)
- From each of the above groups it is possible to determine the most likely causes and commence an appropriate strategy of investigation and treatment.
- In many patients investigation and treatment should proceed simultaneously.
- There are two physical signs that are particularly useful in distinguishing psychogenic coma from an organic cause of coma

1. BELL'S PHENOMENON

- This is a normal reflex that is lost with decreasing consciousness.
- When the eye closes, the eye rolls upwards and inwards.
- This reflex is lost with a reduced level of consciousness.
- A patient with an **organic cause of coma** will have lost this reflex, so the eye will not move. The eyelids will close slowly and incompletely.
- A patient with a **psychogenic cause of coma** will have an intact reflex, so the eye will roll upwards. Also, the eyelids will close at a normal speed and completely.

2. THE HAND DROP TEST.

- With the patient lying supine, lift the patient's hand above the face and allow it to drop onto the face.
- A patient with **psychogenic cause of coma** will guide the hand to fall away from the face, while a patient with an **organic cause of coma** will allow the hand to fall onto the face.

ED MANAGEMENT OF COMA:

- **Bed side test: Blood Glucose**
- **ABC DEFG** approach
- Treat **Hypoxia and Hypotension** to prevent further neurological damage: **15 l/min O₂ via a well fitting non-re-breath mask.**
- **Urgent blood gas analysis** and calculation of the **anion gap.**
- **A serum lactate:** degree of tissue hypo-perfusion and is useful as a marker in sepsis
- In cases of non-traumatic coma, the attending doctor should consider specific treatment with **Naloxone, Flumazenil, Thiamine and Glucose.**
- **Flumazenil and Naloxone:** if overdose of benzodiazepines or opiates.
- Wernickes encephalopathy is a rare cause of coma, however indiscriminate infusion of glucose in (thiamine deficient) alcoholics can precipitate further acute neurological damage. In consequence, all malnourished and alcoholic patients in coma should receive **100 mg thiamine slowly over 5 minutes prior to the administration of glucose.**
- **Surgical evacuation** of cerebellar haematomas is proven to improve outcome; surgical evacuation of intracerebral haematomas is not.
- Coma following cardiac arrest is not of itself an indication to withdraw therapy. All patients who present in coma with pyrexia should receive broad spectrum antibiotic therapy urgently.

CHAPTER 6. ABDOMINAL PAIN

I. GENERAL APPROACH

• AETIOLOGY OF ABDOMINAL PAIN

- o **Acute abdominal pain** is normally thought of as pain of less than one-week duration.
- o Almost half of all patients who present to the ED will ultimately be classified as having non-specific pain.
- o Pain associated with the abdomen falls in to one of three types and it is important to identify this at the earliest possible stage.
 - Visceral Pain
 - Parietal (somatic) pain
 - Referred pain
- o To aid initial diagnosis, investigation and management, abdominal pain can be categorised into one of the five areas:

Gastrointestinal	<ul style="list-style-type: none"> • Esophagitis • Gastritis • PUD • Gallstones • Pancreatitis • Acute liver failure • Bowel obstruction 	<ul style="list-style-type: none"> • Diverticular Disease • IBS • Ischaemic bowel • Incarcerated hernia • Gastroenteritis • Constipation
Gynaecological	<ul style="list-style-type: none"> • Ectopic Pregnancy • PID • Ruptured Ovarian cyst 	
Urological	<ul style="list-style-type: none"> • Renal colic • Pyelonephritis • UTI • Testicular Torsion • Epididymo-orchitis 	
Medical	<ul style="list-style-type: none"> • AMI • DKA • Pneumonia • Mesenteric Adenitis • hypercalcaemia 	
Vascular	<ul style="list-style-type: none"> • AAA • Mesenteric Ischaemia 	

DIFFERENTIAL DIAGNOSIS BASED ON AGE GROUP

Age	Differential diagnosis	
Infants	<ul style="list-style-type: none"> • Meconium ileus • Hypertrophic pyloric stenosis • Intussusception • Appendicitis 	<ul style="list-style-type: none"> • Hernia • Volvulus • Testicular torsion
Adolescents	<ul style="list-style-type: none"> • Appendicitis • Testicular torsion 	<ul style="list-style-type: none"> • Epididymo-orchitis • Ectopic pregnancy
Elderly	<ul style="list-style-type: none"> • Aortic aneurysm • Urinary retention 	<ul style="list-style-type: none"> • Mesenteric infarction • Acute cholecystitis

CLINICAL FEATURES SUGGESTING PARTICULAR CAUSES OF ABDOMINAL PAIN

CLINICAL FEATURES	DIFFERENTIAL DIAGNOSIS
<i>Abdominal pain in patients with atherosclerotic disease/AF.</i>	<ul style="list-style-type: none"> • Aortic aneurysm • Mesenteric infarction (embolic or thrombotic)
<i>Abdominal pain out of proportion to clinical findings.</i>	<ul style="list-style-type: none"> • Aortic aneurysm • Mesenteric infarction • Renal colic
<i>Flank pain radiating to the groin.</i>	<ul style="list-style-type: none"> • Renal colic • Pyelonephritis • Testicular torsion • Aortic aneurysm
<i>Severe abdominal pain radiating through to back.</i>	<ul style="list-style-type: none"> • Aortic aneurysm • Acute cholecystitis • Ascending cholangitis • Acute pancreatitis • Peptic ulcer disease
<i>Abdominal pain associated with shoulder tip pain (due to diaphragmatic irritation).</i>	<ul style="list-style-type: none"> • Ectopic pregnancy • Acute pancreatitis • Acute cholecystitis • Ascending cholangitis • Aortic aneurysm • Bowel perforation
<i>Abdominal pain with collapse or signs of shock.</i>	<ul style="list-style-type: none"> • Aortic aneurysm • Ectopic pregnancy • Massive GI bleed • Myocardial infarction
<i>Abdominal distension</i>	<ul style="list-style-type: none"> • Bowel obstruction • Pregnancy • Ascites • Cancer
<i>Evidence of GI bleeding (haematemesis or melaena).</i>	<ul style="list-style-type: none"> • Peptic ulcer • Diverticular disease • Malignancy • Varices • Angiodysplasia
<i>Abdominal bruising</i>	<ul style="list-style-type: none"> • Trauma • Aortic aneurysm • Acute pancreatitis <ul style="list-style-type: none"> ◦ haemorrhagic fluid collecting in the paracolic gutters (Grey Turner's sign) or around umbilicus (Cullen's sign)
<i>Constipation</i>	<ul style="list-style-type: none"> • Bowel obstruction • Bowel ischaemia • Diverticular disease

CLINICAL ASSESSMENT

- It is vital to take an accurate pain history as this can provide important information.
- There are few key questions to ask about abdominal pain:
 - **Onset**: sudden or gradual
 - **Duration** or recurrence of the pain
 - **Character** or nature of the pain
 - **Location** of the pain
- Furthermore, **associated symptoms** will provide further information on the patients condition:
 - Nausea and vomiting: Pain followed by vomiting does suggest surgical cause
 - Altered bowel habit: Diarrhoea, Constipation, Rectal Bleeding, Melenia
 - Urinary symptoms: Dysuria, Frequency, Haematuria
 - Gynaecological history: LMP, PVB, PVD, Dyspareunia
 - Genito-urinary Medicine: sexual history
- **Abdominal Examination**
 - It is important to **gently palpate** each of the abdominal areas looking at the patients face for signs of pain before proceeding to deep palpation.
 - On examination, note **any tenderness or guarding**, for example, tender to light palpation in the suprapubic area but without signs of guarding.
 - It is important to reassess the abdomen regularly as serial examinations by the same physician may reveal worsening pathology e.g. peritonitis.
 - Do not forget to check for **organomegaly** as this is an important part of the abdominal examination.

INVESTIGATION STRATEGIES

- Investigations in the ED **MUST** include the following simple tests:
 - **Urinalysis** This should be performed in **ALL** patients with abdominal pain and if significant infection suspected an urgent gram stain should be arranged
 - **Urinary hCG** This **MUST** be performed in all females of child-bearing age to rule out ectopic pregnancy
 - **BM Stix** Do not **FORGET** this, as DKA can present with abdominal pain
 - **Blood Tests**: ABG, WCC, CRP, U&E, LFT, Amylase, G&S±Cross Match
 - **Imaging Tests**: ECG, PFA, CXR (Erect), US, CT
 - **Special/Later Tests**: The following tests and imaging are normally performed outside of the ED: further blood tests, Ba Enema, Endoscopy, Angiogram, MRI...

ED MANAGEMENT OF ACUTE ABDOMEN

- **Resuscitate** if signs of sepsis or haemodynamic instability are shown, furthermore if there are any concerns or patient is unwell, discuss with ED senior.
- **Morphine IV titrated to effect**: There is no evidence that opiates mask the signs of peritonism or lead to a delay in diagnosis. Analgesia should never be withheld until the patient has seen the surgeon.
 - *Tramadol is sometimes prescribed for patients with abdominal pain. However, it is difficult to titrate and morphine remains the analgesic of choice*
 - *There is no evidence for anti-spasmodics like **Buscopan** in the management of acute pain.*
- **IV anti-Emetic: Metoclopramide** theoretically increases gastric emptying so **cyclizine** has been favoured in the past although there is little evidence to support this.

- **Anti-pyretic** (IV paracetamol if necessary).
- **Nasogastric tube** if a bowel obstruction is present.
- **Urinary catheter** if the patient is unwell or peritonitis is suspected.
- **Broad spectrum IV antibiotics** if there are signs of sepsis or peritonitis (local policies vary but normally a 2nd generation cephalosporin +/- metronidazole).
- **Keep nil by mouth** and **give IV fluids** (normal saline).
- **Refer to surgical team.**

ADMIT/DISCHARGE DECISION MAKING

- If there is no evidence of a significant surgical pathology, the patient is pain free and has a normal examination then it is reasonable to discharge the patient home with clear advice to **return to the department if their pain recurs or they have any concerns.**
- It may also be appropriate to arrange a review **12-24 hours later.** Patients with suspected biliary colic who have pain that settles can often undergo USS followed by discharge or be brought back the next day for an USS. Elderly patients and those with significant co-morbidity should be admitted as they are at much higher risk of significant pathology.
- It is not uncommon to reassess a patient who initially required morphine for pain to find them pain free with a soft abdomen. Remember these patients have had IV opiates and their pain may recur once this wears off.
- In general, they need to be admitted for observation and possible further investigations.

II. APPENDICITIS

Clinical assessment:

- **History**
 - The classical history in acute appendicitis is that of **initial colicky central abdominal pain** that moves after 6-12 hours to the right iliac fossa where it is constant in nature.
 - This classic history is only normally present in half of the patients that present to the ED with appendicitis.
 - Other common symptoms include anorexia (which tends to be present in 80% of patients with appendicitis), nausea, vomiting (which starts after the pain) and constipation.
 - Appendicitis often presents with an atypical history particularly in the elderly, children and pregnant patients and can make these a very difficult diagnostic group, where the diagnosis may be made late and the risk of perforation is higher. The classic migrating of pain of appendicitis has a sensitivity and specificity of around 80%.
 - **Why does the pain of appendicitis move?**
 - The appendix is *innervated by the autonomic nervous supply to the mid-gut.*
 - Inflammation in the appendix activates **afferent sympathetic fibres**, which enter the spinal cord **at T10** and resulting in referred colicky pain to the peri-umbilical area.
 - Eventually inflammation in the appendix will irritate the surrounding parietal peritoneum, which is innervated by the intercostal nerves resulting in constant local pain in the right iliac fossa.
- **Examination**
 - The patient can look flushed, dehydrated and have a furred dry tongue with **fetor oris.**
 - They may also have a fever and slight tachycardia.

- o Patients are in pain, want to lie still and have tenderness in the right iliac fossa, maximal over McBurney's point which lies 1/3 of the way along an imaginary line from the anterior superior iliac spine to the umbilicus and indicates where the inflamed appendix normally lies.
- o Signs of localised peritonism in a patient with suspected appendicitis:
 - **Direct tenderness** - press in RIF and patient experiences pain
 - **Guarding** - due to voluntary or involuntary contraction of abdominal muscles
 - **Rigidity** - due to reflex spasm of abdominal wall muscles
 - **Rebound** - press enough to depress peritoneum in the RIF for 30s, suddenly remove hand, patient experiences rebound pain
 - **Rovings sign** - press deeply in the LIF for 30s, release suddenly, patient experiences rebound tenderness in RIF
 - **Psoas sign** - ask patient to lift flexed thigh against your hand placed just above the knee, patient experiences pain in RIF.
- o Appendicitis largely remains a clinical diagnosis based upon piecing together the history and examination
- o **McBurney's point** is defined as being the point that lies one-third of the distance from the anterior superior iliac spine to the umbilicus. It roughly corresponds with the most common position of the attachment of the base of the appendix to the caecum.

DIFFERENTIAL DIAGNOSIS:

GASTRO-INTESTINAL	GYNAECOLOGICAL	UROLOGICAL
Terminal ileitis	Ectopic pregnancy	Renal colic
Mesenteric Adenitis	PID	Urinary Tract Infection
Meckels Diverticulitis	Ruptured ovarian cyst	Pylonephritis
Diverticulitis	Ovarian torsion	
Acute cholecystitis		
Gastroenteritis		
Bowel obstruction		
Non-specific abdominal pain		

INVESTIGATION STRATEGIES

- o Urinalysis/ Urinary beta hCG
- o FBCC-reactive protein
- o Plain abdominal x-ray: there is no role for plain films in patients with RIF pain, unless to look for another diagnosis (such as obstruction).
- **Migration of pain, RIF rigidity and guarding with raised inflammatory markers** in combination strongly suggest appendicitis.

ALVARADO SCORE

ALVARADO score = MANTRELS (TL=2)		
M	Migration of pain to RIF	1
A	Anorexia	1
N	Nausea and vomiting	1
T	Tenderness in RIF	2
R	Rebound pain	1
E	Elevated temperature	1
L	Leukocytosis	2
S	shift of WBC to left	1
Total		10

ADDITIONAL IMAGING INVESTIGATIONS• **USS**

- o It has an overall accuracy of about **90%** (sensitivity 84% and specificity 88%) but is very operator dependent.
- o *An USS can rule in appendicitis but cannot rule it out*, i.e. in the presence of a normal scan the patient will still need to be closely observed.
- o USS is of particular value in trying to identify other pathologies, especially in women of childbearing age, when the diagnosis may unclear.
- o It is also of benefit in patients with atypical signs, such as the elderly, children or pregnant patients.

• **CT**

- o CT has a greater overall accuracy of **94%** (sensitivity 94%, specificity 95%) in diagnosing appendicitis compared to USS.
- o However, CT is costly, may not be readily available and can result in significant radiation exposure to the patients.
- o *Similarly to USS, CT can rule in but not rule out appendicitis.*
- o CT may be better at identifying other pathologies than USS.
- o If a patient is felt to have a high likelihood of appendicitis then unnecessary imaging should not delay theatre, remember the mortality and morbidity in appendicitis is higher if the appendix perforates.
- o Difficult diagnostic groups in patients with suspected appendicitis:

Group	Difficulty
Children	Atypical symptoms and signs can lead to late presentation
Elderly	Atypical symptoms and signs can lead to late presentation and 3x increased perforation rate
Pregnant patients	Abnormal position of the appendix due to pregnant uterus can cause atypical signs, perforation associated with foetal mortality
Abnormal positioning of the appendix	Atypical site of pain, e.g. with pelvic appendix
Women of child-bearing age	Extensive differential diagnosis including tubo-ovarian pathologies, higher rates of negative appendicectomies

ED MANAGEMENT OF APPENDICITIS**1. Resuscitate:** if dehydrated or signs of sepsis

- **Oxygen** (high flow, non-rebreather mask)
- **Intravenous access x2**
- **IV normal saline 1-2 litres** then reassess
- **Give immediate antibiotics** if patient has signs of septicaemia or generalised peritonitis (cephalosporin and metronidazole)

2. Analgesia

- **Morphine IV** titrated to effect with **IV anti-emetic**.

3. Keep nil by mouth**4. Involve surgical team****5. Appendicetomy**

III. ACUTE PANCREATITIS

- Acute pancreatitis is a relatively common and serious cause of acute abdominal pain.
- It is acute inflammation of the pancreas that results in the release of enzymes that cause autodigestion of the organ.
- The commonest causes of acute pancreatitis are **Gallstones and Alcohol**. Many cases are also idiopathic. The mnemonic '**I GET SMASHED**' is a useful memory aid for remembering the various causes:
 - **I**: Idiopathic
 - **G**: Gallstones
 - **E**: Ethanol
 - **T**: Trauma
 - **S**: Steroids
 - **M**: Mumps
 - **A**: Autoimmune
 - **S**: Scorpion stings
 - **H**: Hyperlipidaemia/hypercalcaemia
 - **E**: ERCP
 - **D**: Drugs
- **Clinical features of acute pancreatitis include:**
 - Epigastric pain (can be severe)
 - Nausea and vomiting
 - Referral to **T6-T10 dermatomes** (or shoulder tip via phrenic nerve if diaphragmatic irritation)
 - Pyrexia/sepsis
 - Epigastric tenderness
 - Jaundice
 - **Gray-Turner sign** (ecchymosis of the flank)
 - **Cullen sign** (ecchymosis of peri-umbilical area)

Cullen's sign



Turner's sign



Figure 1.6.1. Cullen and Gray-Turner signs

- **Signs of tetany**, such as fasciculations, twitching and a **positive Trousseau's or Chvostek's test**, should also be looked for since hypocalcaemia can develop secondary to intra-abdominal fat necrosis.

38 FRCEM Intermediate

• INVESTIGATIONS IN THE ED SHOULD INCLUDE:

- Blood glucose (or BM stick testing)
- Full blood count (raised white cell count common)
- Urea and electrolytes, calcium, liver function tests
- Coagulation screen, Serum amylase (> 5 times normal limit)
- ECG
- Arterial blood gas
- Abdominal X-ray

- **Amylase** is an enzyme that catalyses the hydrolysis of starch into sugars. It is produced by the pancreas and the salivary glands. Amylase typically rises in acute pancreatitis within 6-12 hours of the onset of the attack.

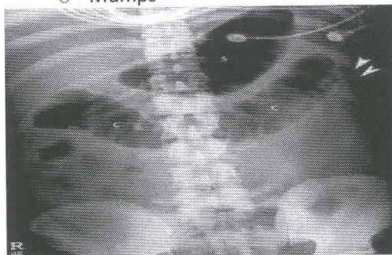
• It also rises in several other conditions including:

- Renal failure
- Ectopic pregnancy
- Diabetic ketoacidosis
- Perforated duodenal ulcer
- Mesenteric ischaemia / infarction
- Pancreatic carcinoma
- Burns
- Mumps

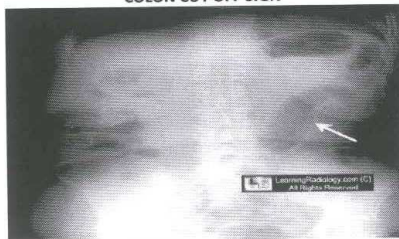
Radiograph of Acute Pancreatitis:

- The abdominal radiograph is not diagnostic and frequently normal or may demonstrate:
 - *Sentinel loop*
 - *Colon cut off sign*
 - *Diffuse ileus*
 - *Pleural effusion*

Sentinel loop: a focal dilated jejunal loop in the LUQ



COLON CUT OFF SIGN



SENTINEL LOOP SIGN

• RISK ASSESSMENT

1. RANSON CRITERIA

AT ADMISSION	AT 48 HOURS
Age > 55	Calcium < 8.0 mg
WCC > 16	Haematocrit fall $> 10\%$
Glucose > 11	PO ₂ < 60
AST > 250	Urea increased by 1.8
LDH > 350	Base excess 6 L
	Initial Assessment
	Clinical impression of severity
	Body Mass Index > 30
	Pleural effusion on chest radiograph

2. GLASGOW PROGNOSTIC CRITERIA

- The Glasgow system is a simple prognostic system that uses age, and 7 laboratory values collected during the first 48 hours following admission for pancreatitis, to predict severe pancreatitis. It is applicable to both biliary and alcoholic pancreatitis.
- A point is assigned if a certain breakpoint is met at any time during that 48-hour period.
- 3 or more of the following detected within 48 hours of admission is suggestive of severe pancreatitis and may require ITU input:
- **Mnemonic "PANCREAS"**
 - PaO₂ <8kPa
 - Age >55yrs
 - Neutrophilia: WCC >15×10⁹/L
 - Calcium <2mmol/L (normal: 2.12mmol-2.65mmol/L)
 - Renal function: urea >16mmol/L (normal: 2.5-6.7mmol/L)
 - Enzymes: LDH >600iU/L (normal: 70-250iU/L); AST >200iU/L (normal: 5-35iU/L)
 - Albumin <32g/L (serum)
 - Sugar: blood glucose >10mmol/L
- **A score ≥ 3 indicates Acute Severe Pancreatitis**
- **A score = 2 indicates Acute Moderate Pancreatitis**
- **A score < 2 indicates Acute Mild Pancreatitis**

• COMPLICATIONS

<i>Early complications include:</i>	<i>Late complications include:</i>
<ul style="list-style-type: none"> ○ Severe sepsis and circulatory shock ○ Acute renal failure ○ Disseminated intravascular coagulation ○ Hypocalcaemia ○ Acute respiratory distress syndrome ○ Pancreatic encephalopathy ○ Multi-organ failure 	<ul style="list-style-type: none"> ○ Pancreatic pseudo-cyst ○ Pancreatic abscess ○ Insulin dependant diabetes mellitus ○ Chronic pancreatitis

• ED MANAGEMENT OF ACUTE PANCREATITIS

- Aim for SaO₂ >95% and a urine output of >0.5 ml/Kg.
- Resuscitate if dehydrated or signs of sepsis
- Oxygen- high flow through variable delivery mask
- Intravenous access x2
- IV Normal Saline 1-2L then reassess (may require several litres of fluid resuscitation)
- Analgesia opiate titrated to effect (Tramadol); avoid Morphine
- Anti-Emetic
- Keep nil by mouth
- NG tube only if there is evidence of an ileus
- Urinary catheter and hourly urine volumes
- IV broad spectrum antibiotics only if signs of sepsis
- Surgical referral: Involve surgical team and admit ALL patients with suspected pancreatitis

IV. BOWEL OBSTRUCTION

INTRODUCTION

- Intestinal obstruction can be classified in several different ways, most traditionally into small and large bowel obstruction.
- Bowel obstruction carries a high morbidity and mortality if managed incorrectly.

CAUSES OF INTESTINAL OBSTRUCTION

COLON	
<ul style="list-style-type: none"> • Tumors (usually in left colon), • Volvulus of sigmoid or cecum, • Diverticulitis (usually in sigmoid), • Fecal impaction, • Hirschsprung disease, • Crohn disease 	
DUODENUM	
Adults	Neonates
<ul style="list-style-type: none"> • Cancer of the duodenum or head of pancreas, • Ulcer disease 	<ul style="list-style-type: none"> • Atresia, • Volvulus, • Bands, • Annular pancreas
JEJUNUM AND ILEUM	
Adults	Neonates
<ul style="list-style-type: none"> • Adhesions (common), • Hernias, • Tumors, • Foreign body, • Meckel diverticulum, • Crohn disease (uncommon), • <i>Ascaris</i> infestation, • Midgut volvulus, • Intussusception by tumor (rare) 	<ul style="list-style-type: none"> • Intussusception • Meconium ileus, • Volvulus of a malrotated gut, • Atresia,

- The most common causes of bowel obstruction are:
 - **Small bowel:** adhesions (60% in UK), hernias, intussusception (paediatric group)
 - **Large bowel:** malignancy (developed countries), volvulus (developing countries)
 - **Functional** (also referred to as paralytic) obstruction is relatively rare as a presentation to the emergency department.
- Functional obstruction results from atony of the intestine and loss of normal peristalsis.
- Atony of the bowel can be localised to a particular segment or generalised throughout the entire bowel.
- Localised atony is thought to result from an abnormality in the myenteric plexus of the bowel wall, whereas more generalised atony probably results from an imbalance in autonomic nerve supply, although there is little direct evidence for this.
- Different terms are often used to describe functional obstruction of the small or large bowel: **paralytic ileus** and **pseudo-obstruction** respectively.

HERNIAS

- Hernias can be classified in the following table:

HERNIA	ANATOMY	INCIDENCE
Indirect inguinal hernia	Bowel passes through inguinal canal via a congenital weakness of the internal inguinal ring	most common
Direct inguinal hernia	Hernia exits abdominal cavity directly through the deep layers of the abdominal wall	uncommon
Femoral hernia	Abdominal contents pass through femoral canal just below inguinal ligament	rare

- Inguinal hernias** are the most common type of hernia in both men and women, the indirect type accounting for 2/3 of cases.
- Almost all femoral hernias occur in women because of the wider bone structure of the female pelvis; however inguinal hernias are still more common in women than femoral hernias.

CLINICAL ASSESSMENT

- History: AMPLE**
 - Classic symptoms of intestinal obstruction are **colicky abdominal pain, abdominal distension, vomiting and constipation**.
 - Vomiting is a late feature with large bowel obstruction.
 - History of Previous abdominal surgery
 - Remember to pay particular attention to the:
 - Reproductive, contraceptive and menstrual history
 - Possibility of pregnancy and its complications
 - Drug history, especially favoured remedies and alcohol consumption
 - Past medical history
 - Severe pain suggests strangulation and developing ischaemia in a closed loop of bowel.
- Severe pain in bowel obstruction suggests complications such as ischaemia or perforation
- Examination**
 - Begin with baseline observations looking for any physiological evidence of dehydration or shock.
 - Adequately expose the patient to examine the abdomen meticulously, looking for surgical scars, peritonism, masses and not forgetting the hernial orifices.
 - It is easy to miss a small femoral hernia in an obese patient.
 - Bowel sounds may be high-pitched / tinkling or absent altogether.
 - Careful attention should also be made to look for other causes of the acute abdomen, in particular a ruptured abdominal aortic aneurysm (**AAA**) or **ectopic pregnancy**.
 - A rectal examination must also be performed**, perhaps demonstrating an empty rectum or obstructing mass, ideally once only by the clinician making the management decisions.
 - Constipation and abdominal distension in the patient with a previous history of bowel surgery are strongly suggestive of intestinal obstruction*
 - The six variables with highest sensitivity for a diagnosis of bowel obstruction were a distended abdomen, increased bowel sounds, history of constipation, previous abdominal surgery, age over 50 years and vomiting.*

INVESTIGATION STRATEGIES

- **General / Basic**
 - **ABG** if signs of sepsis or strangulated bowel
 - **Urinalysis**
 - **ECG**
 - Blood tests: **FBC, U&E, LFT, GLUCOSE, AMYLASE, GROUP&SAVE.**
 - **Clotting screen** if septic or on anticoagulants.
 - **Plain film x-ray erect and supine abdominal x-ray/ Chest x-ray**
- **Specific Imaging**
 - **CT**
 - **Small bowel follow-through**
 - **Water-soluble contrast enema**

RADIOLOGICAL SIGNS OF BOWEL OBSTRUCTION

- If a patient presents with clinical features of obstruction then radiological assessment can be very helpful in determining the level of obstruction, and occasionally the cause.
- There are features visible on a plain abdominal X-ray that may help locate the level of obstruction.
- These are partly determined by a knowledge of small and large bowel anatomy

1. SMALL BOWEL OBSTRUCTION

- **KEY FEATURES OF SBO:**
 - **Dilated small bowel loop >2.5cm in diameter**
 - **The relatively central position** of the small bowel and restriction in dilatation to 5cm also helps to distinguish small from large bowel on plain films.
 - **Presence of valvulae conniventes**, which completely cross the bowel wall. **The taenia coli** of the large bowel are incomplete across the bowel wall.

**PARALYTIC ILEUS**

- Paralytic ileus, also called **pseudo-obstruction**, is one of the major causes of intestinal obstruction in infants and children.
- **Causes of paralytic ileus may include:**
 - Bacteria or viruses that cause intestinal infections (gastroenteritis)
 - Chemical, electrolyte, or mineral imbalances (such as decreased potassium level)
 - Abdominal surgery
 - Decreased blood supply to the intestines
 - Infections inside the abdomen, such as appendicitis
 - Kidney or lung disease
 - Use of certain medicines, especially narcotics

SENTINEL LOOP

- Intra-abdominal inflammation, such as with pancreatitis, can lead to a localized ileus.
- This may appear as a single loop of dilated bowel known as a 'sentinel loop.'

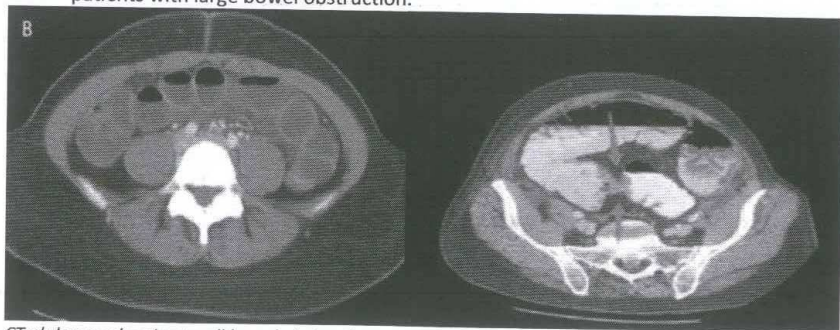
2. LARGE BOWEL OBSTRUCTION

- The most common causes of large bowel obstruction are **colo-rectal carcinoma** and **diverticular strictures**. Less common causes are **hernias** or **volvulus** (twisting of the bowel on its mesentery). Adhesions do not commonly cause large bowel obstruction.
- Radiological appearances of large bowel obstruction differ from those of small bowel obstruction, however, with large bowel obstruction there is often co-existing small bowel dilatation proximally.
- Abdominal X-ray cannot reliably differentiate mechanical obstruction from pseudo-obstruction
- **KEY FEATURES OF LBO:**
 - Dilatation of the caecum **>9cm** is abnormal
 - Dilatation of any other part of the colon **>6cm** is abnormal
 - Abdominal X-ray may demonstrate the level of obstruction



• CT Scan

- It is the most useful in differentiating the specific cause and location of mechanical obstruction. It is the imaging modality of choice for the investigation of patients with inconclusive plain films for complete or high-grade small bowel obstruction and for patients with large bowel obstruction.



CT abdomen showing small bowel obstruction

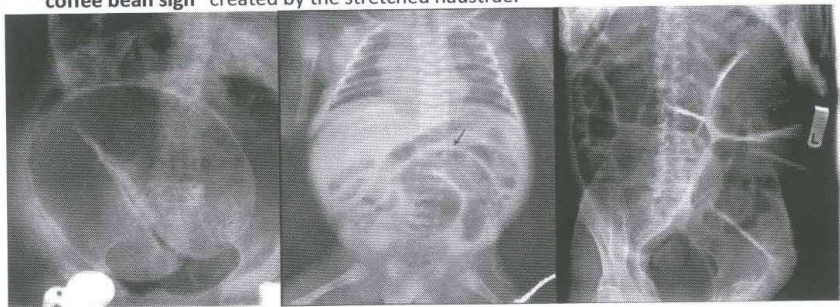
CT abdo/pelvis showing large bowel obstruction with apple core constricting lesion

ED MANAGEMENT OF BOWEL OBSTRUCTION**A. GENERAL ED MANAGEMENT**

- o **O2 and fluid resuscitation** if the patient is haemodynamically unstable
- o **Insert an IV cannula** (taking and sending blood as mentioned above)
- o **Move the patient to an appropriate area of the department and involve an ED senior in their management.**
- o **Start an IVI of 0.9% Saline/ Titrate IV analgesia** (morphine) **with an antiemetic**
- o **Insert a naso-gastric tube** and declare the patient **Nil by Mouth (NBM)**
- o **Insert a urinary catheter**
- o **Consider more invasive monitoring if required for accurate fluid resuscitation** (CVP and/or arterial line)
- o **Broad spectrum antibiotics** are commonly administered because of concerns that bacterial translocation may occur in the setting of small bowel obstruction; however, there are no controlled data to support or refute this approach.
- o **REFER TO THE SURGICAL TEAM**

3. SIGMOID VOLVULUS

- Rotation (clockwise = anticlockwise) of section of intestine on its mesentery. Sigmoid volvulus occurs when a redundant portion of sigmoid colon twists around its mesentery.
- The sigmoid is the commonest site of volvulus but it can occur at other sites, especially the caecum (caecal volvulus). It often occurs in elderly or institutionalised patients with a history of chronic constipation.
- Volvulus usually presents with **pain, abdominal distension, vomiting and absolute constipation**, but in elderly or confused patients pain may be surprisingly limited.
- The blood supply is compromised and venous congestion occurs. There is progressive accumulation of gas and bowel fluid proximal to the obstruction and perforation will occur without prompt diagnosis and definitive management.
- Plain abdominal xray will demonstrate a **grossly distended sigmoid colon** with the a “**coffee bean sign**” created by the stretched haustrae.



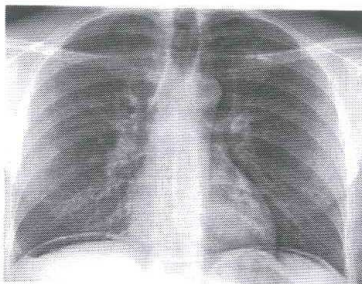
- Once a diagnosis of volvulus has been made, **prompt surgical referral is crucial**.
- The patient may require resuscitation and there can be significant fluid shifts and signs of sepsis due to bowel necrosis.
- A **sigmoidoscope** will successfully decompress the majority of uncomplicated sigmoid volvulus, but definitive surgery may be required if there is evidence of necrosis or perforation.

V. BOWEL PERFORATION

- Bowel perforation is a life-threatening surgical emergency.
- Perforations may affect any part of the GI tract.

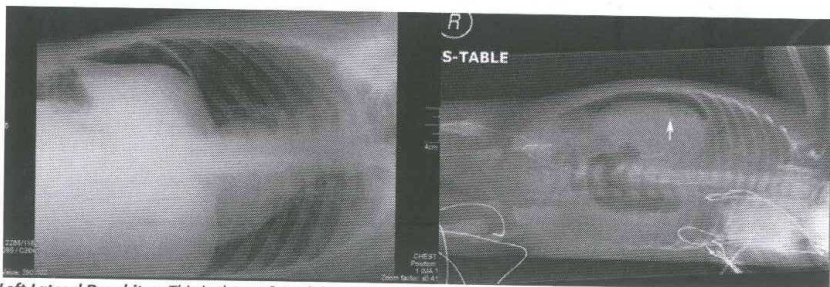
- **CAUSES OF BOWEL PERFORATION**

- Peptic ulcer.
- Appendicitis.
- Diverticulitis.
- Colonic carcinoma.
- Trauma.
- Toxic megacolon.
- Prolonged strangulated bowel.



- **SPECIFIC INVESTIGATIONS FOR BOWEL PERFORATION**

- **Erect CXR**—aims to identify **free intra-peritoneal gas under the diaphragm** due to hollow viscus perforation. Patients should be sat upright for at least 10 minutes before the CXR. **Figures vary but an erect CXR identifies 70–80% of pneumoperitoniums**, so can be used as a rule-in but a not rule-out test.
- **A lateral CXR (left lateral decubitus shoot through)**
 - Has better sensitivity than an anteroposterior film for free air.
 - Patient is allowed to lie down in left lateral decubitus for around 10 minutes, so that **intra-peritoneal air** in the lesser sac can pass through foramen of Winslow into the greater sac and **accumulate between lateral margin of liver and lateral abdominal wall**.



Left Lateral Decubitus: This is the preferred decubitus position. The free intra-peritoneal gas is seen easily because it is contrasted against the liver. Although it is described as a "left lateral decubitus" it is marked with a right marker.

- **Ultrasound**—in trained hands has a greater sensitivity for free intra-peritoneal air than CXR and has the advantage that it can be performed in the resuscitation room.
 - **CT abdomen with contrast**—is the most sensitive investigation and is very useful in patients where there is diagnostic uncertainty.
- **ED MANAGEMENT OF BOWEL PERFORATION**
 - Fluid resuscitation.
 - Intravenous analgesia and anti-emetic.
 - Intravenous broad spectrum antibiotics.
 - Nil by mouth.
 - Urgent surgical referral.

VI. BOWEL ISCHAEMIA/INFARCTION

1. MESENTERIC INFARCTION

- Acute mesenteric infarction may result from an **embolus or thrombosis**.
- It can also be secondary to **profound hypotension** or **mesenteric venous thrombosis**.
- The superior mesenteric artery is most commonly affected by emboli due to the small take-off angle from the aorta and higher flow rate.
- Clinically the patient develops acute severe abdominal pain but often the clinical signs are minimal.
- There may be a history of chronic mesenteric ischaemia ('abdominal angina') with pain after eating, fear of food, and weight loss.
- **Investigations for mesenteric infarction**
 - There is no specific ED investigation to diagnose mesenteric infarction.
 - **Serum lactate** is usually elevated with a **metabolic acidosis**, indicating inadequate perfusion.
 - An **ECG** should be performed looking for evidence of AF and an **echocardiogram** for a mural thrombus.
 - A **plain CT abdomen** may identify another cause for the pain.
 - **CT angiography** is the most useful test in diagnosing mesenteric infarction.

2. ISCHAEMIC COLITIS

- Chronic arterial insufficiency typically occurs at the splenic flexure because it is a watershed territory supplied by the superior and inferior mesenteric arteries.
- Patients report abdominal pain, classically in the left iliac fossa, associated with loose, bloody stools. The patient may have a history of cardiovascular disease and report recurrent episodes of similar pain. Patients should be referred to the surgical team for further investigation and management.
- A **barium enema** may show evidence of '**thumb-printing**' due to submucosal swelling.
- Complications include **stricture formation** and **gangrenous ischaemic colitis**.

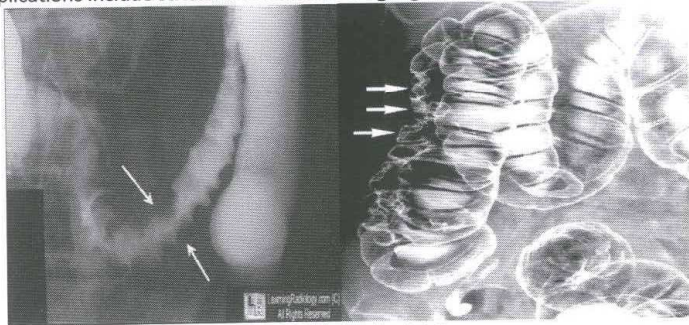


Figure 1.6.2. thumb-print sign

- If a patient has pain out of proportion to clinical findings think of **mesenteric infarction**.
- An **ECG** can be a useful investigation to identify AF and indicate the source of a possible embolus.
- **Lactate** is usually elevated in bowel infarction due to hypoperfusion. An elevated lactate is a non-specific finding and is raised in many causes of acute abdomen. However, a raised lactate gives an indication of the severity of disease.

VII. ABDOMINAL AORTIC ANEURYSM

INTRODUCTION

- An abdominal aortic aneurysm (AAA) is an abnormal dilatation of the aorta.
- The majority are saccular and occur infra-renally.
- Patients over the age of 50, presenting with acute abdominal pain, should always have an abdominal aortic aneurysm considered in their differential diagnosis.
- Rupture usually leads to haemorrhage into the retroperitoneal space.

CLINICAL FEATURES OF AAA

- The classic presentation is **central abdominal and back pain** in a patient with a known aneurysm.
- However, presentation may vary from a PEA arrest to painless, sudden collapse.
- Patients may be mistaken as having renal colic due to the presence of **haematuria** caused by **irritation of the ureter or rupture into the renal artery**.
- Examination may reveal a **tender pulsatile mass**.
- One or both **femoral pulses may be absent**.
- In the obese or elderly, the diagnosis can be particularly challenging and a high index of suspicion should be maintained.

INVESTIGATIONS FOR AAA

- Diagnosis is largely clinical supplemented by the use of **Emergency Ultrasound**.
- Ultrasound is now considered a core skill of Emergency Medicine trainees and one of the main indications is the diagnosis of AAA.
- Emergency ultrasound is a useful rule-in test for identifying an aneurysm but poor for detecting a leak.
- Ultrasound is user dependent and if there is ongoing clinical suspicion of an AAA further imaging is required.
- **CT** is rarely used in the unstable patient with a suspected AAA but maybe used if diagnostic uncertainty persists and the patient is stable.

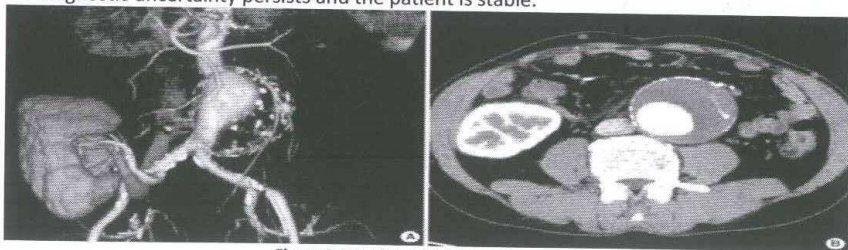


Figure 1.6.3. Abdominal Aortic Aneurysm

ED MANAGEMENT OF AAA

- **ABC** approach.
- **Cautious fluid resuscitation:**
 - Should be instituted, aiming for a **SBP >90 mmHg**.
 - Aggressive fluid resuscitation has been shown to worsen outcome in patients with leaking AAA.
 - Therefore, if the patient is conscious and passing urine, minimal fluid should be given until the aorta is cross-clamped in theatre.
 - The blood bank should be informed and **10 units of blood and 2 units of platelets** should be cross-matched.

VIII. BILIARY TRACT DISORDERS

INTRODUCTION

- The commonest biliary tract disorder presenting to the ED is **Gallstones**.
- Gallstones are precipitants of bile that form in the gallbladder.
- Bile contains cholesterol, bile pigments (from haemoglobin breakdown), and phospholipids.
- The varying concentrations of these components results in 3 main types of stone:
 - **Cholesterol:** large, often solitary stones that account for the majority of UK gallstone disease. Risk factors include increasing age, female sex, obesity, family history, hyperlipidaemia, diabetes, and cystic fibrosis.
 - **Pigmented:** small, dark stones composed of bilirubin and calcium salts. Risk factors for pigmented stones include haemolytic anaemias and cirrhosis.
 - **Mixed:** contain varying amounts of cholesterol, calcium salts, and bilirubin. The calcium salts allow the stones to be seen radiographically. Approximately 10% of gallstones are radio-opaque.

1. BILIARY COLIC

- Biliary colic occurs when a gallstone lodges in the neck of the gallbladder, the cystic duct, or common bile duct.
- The blockage causes increased intraluminal pressure and distension of the gallbladder.
- The gallstone then dislodges and passes out of the biliary tract.
- The patient experiences abdominal pain, often located in the right upper quadrant, associated with nausea and vomiting. The symptoms resolve when the stone passes.
- Patients may suffer recurrent episodes of biliary colic when further stones pass and therefore are diagnosed with chronic cholecystitis.
- An **ultrasound scan** is indicated to confirm the presence of gallstones.
- Blood tests are usually normal.
- If symptoms settle, then outpatient management is appropriate, pending a cholecystectomy.

2. ACUTE CHOLECYSTITIS

- In 95% of acute cholecystitis, a gallstone or biliary sludge becomes impacted at the neck of the gallbladder. Only 5% of patients have no stone; these are usually patients that have been admitted for trauma, burns, or have diabetes.
- **Acalculous acute cholecystitis** has a worse prognosis than those with gallstones.
- The obstructed gallbladder becomes distended, inflamed, and ischaemic.
- Bacteria are able to penetrate the gallbladder wall causing infection.
- Prolonged obstruction may result in a **gallbladder empyema**.
- **Clinical features of acute cholecystitis**
 - **Abdominal pain**—typically located in the right-upper quadrant.
 - Pain is often dull and poorly localized initially due to distension of the gallbladder and stimulation of the visceral peritoneum. As the inflammatory process progresses, inflammatory fluid leaks out stimulating the local parietal peritoneum, which is innervated by intercostal nerves and felt as a sharp, well-localized pain.
 - **Murphy's sign**—this is an indication of local peritonism.

- Deep palpation in the right-upper quadrant, during inspiration, causes pain as the inflamed gallbladder impinges on the palpating hand.
- This causes a sudden inspiratory arrest. The test is only positive if repetition in the left-upper quadrant doesn't cause pain.
- **Jaundice**—may occur if the stone moves and obstructs the common bile duct or if the gallbladder causes compression of the common hepatic duct (**Mirizzi's syndrome**).
- **Specific investigations for acute cholecystitis**
 - **Ultrasound scan**—is the most useful investigation for confirming the diagnosis. It may show gallbladder wall thickening, pericholecystic fluid, and an impacted gallstones.
 - **Amylase or lipase** should be sent to exclude pancreatitis.
 - **Urinary pregnancy test.**
 - **ECG**—to exclude a MI or ACS
 - **CXR**—to exclude pneumonia and look for evidence of air under the diaphragm in a suspected perforation.
- **ED MANAGEMENT OF ACUTE CHOLECYSTITIS**
 - **Fluid resuscitation**—if the patient has signs of sepsis or dehydration
 - **Analgesia**—intravenous morphine titrated to effect
 - **Antibiotics**—usually a 3rd generation cephalosporin but local antibiotic policy should be followed
 - **Nil by mouth**
 - **Urgent surgical review**—surgical options depend on the severity of illness ranging from medical therapy, to endoscopic retrograde cholangiopancreatography (ERCP), to cholecystectomy (open or laparoscopic).

3. CHOLEDOCHOLITHIASIS

- Cholelithiasis is when a gallstone becomes stuck in the common bile duct resulting in jaundice and hepatic damage. Investigations are the same as acute cholecystitis.
- Treatment is removal of the stone via **ERCP**.

4. OBSTRUCTIVE JAUNDICE

- A gallstone in the common bile duct is a cause of post-hepatic jaundice.
- The patient will have dark urine and pale stools.
- **Cholangio-/pancreatic carcinomas** may present in a similar manner but are usually not painful.
- *If the gallbladder is palpable a pancreatic carcinoma is the more likely diagnosis (**Courvoisier's law**: 'In the presence of jaundice, if the gallbladder is palpable, the cause is unlikely to be a stone').*

5. GALLSTONE ILEUS

- Prolonged obstruction and inflammation of the gallbladder may result in a fistula developing between the gallbladder and the duodenum.
- The gallstone can then enter the GI tract and obstruct the terminal ileum.
- An abdominal X-ray may show air in the biliary tree and evidence of small bowel obstruction.
- Patients should be resuscitated and referred urgently for surgical review.

6. ASCENDING CHOLANGITIS

- Occurs when the common bile duct becomes infected, often secondary to a stone in the common bile duct (choledocholithiasis) that has caused chronic bile stasis.
- The classic presentation of ascending cholangitis is with **Charcot's triad**:
 - Jaundice
 - Fever (usually with rigors)
 - Right upper quadrant pain
- Ascending cholangitis is a potentially life-threatening medical emergency and patients are frequently septic.
- 10-20% of patients present with the additional features of altered **mental status** and **hypotension** secondary to septic shock.
- When the two additional features are present in addition to Charcot's triad the patient is said to have the **Reynold's Pentad**.
- The treatment of ascending cholangitis is **urgent biliary drainage**.
- Generally speaking Murphy's sign is usually positive in acute cholecystitis but negative in biliary colic and ascending cholangitis.
- The **white cell count** and **CRP** are usually elevated in ascending cholangitis.
- Jaundice** is often present and **ALP** and **bilirubin** levels can be markedly elevated.
- There is a significant amount of overlap between the presentation of biliary colic, acute cholecystitis and ascending cholangitis and the following table helps to differentiate between these diagnoses:

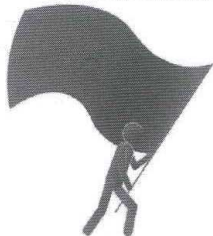
	BILIARY COLIC	ACUTE CHOLECYSTITIS	ASCENDING CHOLANGITIS
Pain duration	< 12 hours	> 12 hours	Variable
Fever	Absent	Present	Present
Murphy's sign	Negative	Positive	Negative
WCC & CRP	Normal	Elevated	Elevated
AST, ALT & ALP	Normal	Normal or mildly elevated	Elevated
Bilirubin	Normal	Normal or mildly elevated	Elevated

CHAPTER 7. ACUTE BACK PAIN

I. GENERAL APPROACH OF BACK PAIN IN ED

Clinical assessment

- **History**
 - Provocative and Palliative factors
 - Quality of pain
 - Radiation
 - Severity and Systemic Symptoms
 - Timing
- **Red flag symptoms indicating possible serious spinal pathology**



- Onset at age <20 or >55
- Non-mechanical pain (i.e. unrelated to time or activity), especially if constant and worsening and pain at night
- Thoracic pain
- Previous history of carcinoma
- Fever, night sweats, weight loss
- Widespread neurological symptoms especially sphincter disturbance
- Structural spinal deformity

- **Examination**
 - The examination of the patient with low back pain may commence with a look, feel, move musculoskeletal approach:
 - Look for stance, deformity, scars etc.
 - Feel for tenderness
 - Check the range of movement
 - It will also include an examination of the nerve supply to the lower limb and perineum.
- **Neurological examination**
 - Each muscle is normally supplied by more than one nerve root, though one nerve root may be dominant.
 - If the patient has any loss of sensation or parasthesiae, it is important to determine whether these are confined to one dermatome or whether they have a different (e.g. stocking) distribution. It is important to test sensation within each dermatome.
 - The neurological examination finishes with examination of the tendon reflexes.
 - While the patient is lying on their back for the neurological examination, it is a good time to test for the ability to straight leg raise. Many patients with back pain will have pain radiating to the buttock or leg but in true sciatica (**the commonest nerve root lesions are L5 and S1**), the pain radiates below the ankle. Some patients with nerve root problems only have pain in the leg and do not have back pain. Patients will have pain if the sciatic nerve is stretched. This occurs on straight leg raising.
 - Dorsiflexion of the foot while the leg is raised will exacerbate the pain (**sciatic stretch test**). In a nerve root lesion the patient will have parasthesiae localised to the dermatome of the involved nerve root and will have altered sensation in the same dermatome. When taking the history from a patient with back pain, it is very important to enquire about **urinary symptoms as retention of urine or incontinence** may indicate involvement of the nerve roots supplying the bladder.

52 FRCEM Intermediate

- o In this case, it is essential to do a **rectal examination**: Examine for the rectal tone and the ability to contract the anal sphincter and while doing the examination note any constipation (which may also cause urinary symptoms) and (in men) feel the prostate (Ca Prostate frequently metastasises to bone and may be a cause of back pain).
- **Sacral Dermatomes**
 - o Abdominal examination is frequently indicated to exclude intra-abdominal causes of back pain (e.g. **ruptured aortic aneurism, retroperitoneal perforation of the colon**).
- **Dermatomes and Myotomes in the lower limb**

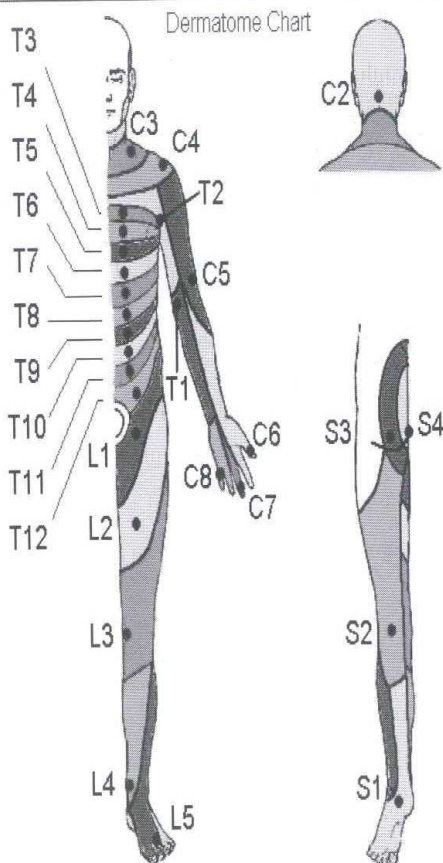
DERMATOMES

C5	Area over the deltoid
C6	Thumb
C7	Middle finger
C8	Little finger
T4	Nipple line
T8	Xiphisternum
T10	Umbilicus
T12	Symphysis pubis
L4	Medial aspect calf
L5	Webspace 1 st and 2 nd toes
S1	lateral border foot
S3	Ischial tuberosity
S4-S5	Perianal region

MYOTOMES

C5	Shoulder abduction
C5, C6	Elbow flexion
	Elbow extension
C7	Wrist extension
	Finger extension
C8	Wrist flexion
	Finger flexion
T1	Finger abduction
L1, L2	Hip flexion
L5, S1	Hip extension
L3, L4	Knee extension
L5, S1	Knee flexion
L4	Ankle dorsiflexion
S1, S2	Ankle plantarflexion
L5	Great toe extension
S1	Great toe flexion

Dermatome Chart



REFLEXES

S1, S2	Ankle
L2, L3, L4	Knee
C5, C6	Biceps
C7,8	Triceps
S3-S4	Anal Wink

DIFFERENTIAL DIAGNOSIS

- o Some of the numerous causes of back pain are listed below.
- o The most important issue for emergency physicians is to be able to differentiate serious from less serious causes and, in particular, to be able to recognise **cauda equina compression**.

CAUSES OF LOW BACK PAIN

Structural	<ul style="list-style-type: none"> • Mechanical or non-specific • Facet joint arthritis or dysfunction • Prolapsed intervertebral disc • Annular tear of intervertebral disc • Spondylolysis or spondylolisthesis • Spinal stenosis
Neoplasm	<ul style="list-style-type: none"> • Primary or secondary including multiple myeloma
Infection	<ul style="list-style-type: none"> • Discitis • Osteomyelitis • Paraspinal abscess
Referred pain to spine from	<ul style="list-style-type: none"> • Major viscera • Retroperitoneal structures • Aorta • Hip
Inflammatory	<ul style="list-style-type: none"> • Spondyloarthropathies • Sacroiliitis or sacroiliac dysfunction
Metabolic	<ul style="list-style-type: none"> • Osteoporotic vertebral collapse • Pagets disease • Osteomalacea • Hyperparathyroidism

POSSIBLE DIAGNOSIS	RED FLAGS
Vertebral fracture	<ul style="list-style-type: none"> • History of trauma (this may be minimal in the elderly or those with osteoporosis). • Prolonged steroid use.
Tumour	<ul style="list-style-type: none"> • Age <20 or >50. • History of malignancy. • Non-mechanical pain. • Thoracic pain. • Systemically unwell. • Weight loss.
Spinal infection	<ul style="list-style-type: none"> • Fever. • Systemically unwell. • Recent bacterial infection. • Non-mechanical pain. • Pain worse at night. • IV Drug Users. • Immunosuppression. • HIV.
Cauda equina syndrome	<ul style="list-style-type: none"> • Saddle anaesthesia. • Bladder or bowel dysfunction. • Gait disturbance. • Motor weakness (widespread or progressive) • Bilateral sciatica.

AAA	<ul style="list-style-type: none"> • Systemically unwell. • Cardiovascular compromise. • Pulsatile abdominal mass.
Inflammatory rheumatic disease (e.g. ankylosing spondylitis)	<ul style="list-style-type: none"> • Age <20. • Structural deformity of the spine. • Systemically unwell.

INVESTIGATION STRATEGIES

- o No investigation is required for the vast majority of patients with non-specific back pain.
- o Red flag symptoms or signs suggestive of cauda equina syndrome will mandate **urgent MRI scanning**.
- o **Imaging** of patients with non-specific back pain and no red flag symptoms or signs is unhelpful. Many patients with spinal pathology may have normal plain X-rays and, conversely, many patients with no back pain may have X-ray abnormalities (particularly degenerative disease).
- o Similarly, many patients with no back pain have abnormal MRI scans.
- o **Blood tests** may be useful if one suspects infection or metabolic problems but are not necessary as screening investigations for patients with no pointers to those problems.

ED MANAGEMENT OF BACK PAIN

- **Symptomatic treatment of acute musculoskeletal lower back pain**
 - o Analgesia.
 - o Muscle relaxants.
 - o Patients should be advised to stay active.
 - o Physiotherapy.
 - o **Other treatments that have been investigated for low back pain are:**
 - Traction, Massage, Antidepressants.
 - Local heat, Acupuncture
 - Individual patient education for low back pain.
 - Spinal manipulative therapy.
 - Exercise therapy, Lumbar supports
 - Strong opiates (eg oramorph)
 - o Note that the above evidence largely relates to acute back pain. The results for chronic and subacute back pain may be different.
- **Treatment of Cauda Equina Syndrome**
 - o Urgent referral is required once the diagnosis has been made on MRI scanning.
- **Treatment of sciatica**
 - o Epidural
 - o Surgical discectomy
 - o Microdiscectomy
- **Treatment of vertebral compression fractures**
 - o **Osteoporosis** will also need to be investigated and managed.
 - o Postmenopausal women with an initial fracture are at much greater risk of subsequent fractures so this is very important and may help to prevent a future attendance with a hip fracture.
- **Treatment of metastatic disease**
 - o Patients with bone metastases and patients at high risk of developing bone metastases should be given information explaining what to do and who to contact if they develop

symptoms of spinal metastases or spinal cord compression or if their symptoms progress while waiting for investigation.

- o **Spinal cord compression** is an oncological emergency and treatment should be started within 24 hours. Most patients will be given **steroids** and will need **radiotherapy or surgery**. Patients with a risk of spinal instability should be nursed flat in neutral alignment.

II. CAUDA EQUINA SYNDROME

- Low back pain affects millions of people every year, and in most cases, it improves without surgery. But severe back pain can be a symptom of a serious condition that is not well known and is often misdiagnosed.
- **Cauda equina syndrome (CES)** occurs when the nerve roots of the cauda equina are compressed and disrupt motor and sensory function to the lower extremities and bladder.
- Patients with this syndrome are often admitted to the hospital as a medical emergency.
- CES can lead to **incontinence** and even **permanent paralysis**.
- The collection of nerves at the end of the spinal cord is known as the **cauda equina**, due to its resemblance to a horse's tail.
- The spinal cord ends at the upper portion of the lumbar (lower back) spine. The individual nerve roots at the end of the spinal cord that provide motor and sensory function to the legs and the bladder continue along in the spinal canal.
- The cauda equina is the continuation of these nerve roots in the lumbar region. These nerves send and receive messages to and from the lower limbs and pelvic organs.
- **INCIDENCE**
 - o CES is not related to gender or race. It occurs primarily in adults, although trauma-related CES can affect people of all ages.
 - o CES affects a very small percentage of patients that have undergone surgery for lumbar herniated disc.
- **CAUSES**
 - o CES most commonly results from a **massive herniated disc in the lumbar region**.
 - A single excessive strain or injury may cause a herniated disc.
 - However, disc material degenerates naturally as you age, and the ligaments that hold it in place begin to weaken.
 - As this degeneration progresses, a relatively minor strain or twisting movement can cause a disc to rupture.
 - o **The following are other potential causes of CES:**
 - Spinal lesions and tumors
 - Spinal infections or inflammation
 - Lumbar spinal stenosis
 - Violent injuries to the lower back (gunshots, falls, auto accidents)
 - Birth abnormalities
 - Spinal arteriovenous malformations (AVMs)
 - Spinal hemorrhages (subarachnoid, subdural, epidural)
 - Postoperative lumbar spine surgery complications
 - Spinal anesthesia

SYMPTOMS AND DIAGNOSIS

- CES symptoms mimic those of other conditions. Its symptoms may vary in intensity and evolve slowly over time.
- CES is accompanied by a range of symptoms, the severity of which depend on the degree of compression and the precise nerve roots that are being compressed.
- Besides a herniated disc, other conditions with similar symptoms to CES include:
 - **Peripheral nerve disorder,**
 - **Conus medullaris syndrome,**
 - **Lumbosacral plexopathy:** spinal cord compression, and irritation or compression of the nerves after they exit the spinal column and travel through the pelvis.
- Patients with back pain should be aware of the following "**red flag**" symptoms that may indicate CES:
 - *Severe low back pain*
 - *Motor weakness, sensory loss, or pain in one, or more commonly both legs*
 - *Saddle anesthesia (unable to feel anything in the body areas that sit on a saddle)*
 - *Recent onset of bladder dysfunction (such as urinary retention or incontinence)*
 - *Recent onset of bowel incontinence*
 - *Sensory abnormalities in the bladder or rectum*
 - *Recent onset of sexual dysfunction*
 - *A loss of reflexes in the extremities*
- **Medical history implications:**
 - Recent violent injury to the back
 - Recent lumbar spine surgery
 - A history of cancer
 - Recent severe infection
- **The following tests may be helpful in diagnosing CES:**
 - **Magnetic resonance imaging (MRI)**
 - **Myelogram:** can show displacement on the spinal cord or spinal nerves due to herniated discs, bone spurs, tumors, etc.
- **TREATMENT:**
 - Once the diagnosis of CES is made, and the etiology established, **urgent surgery is usually the treatment of choice.**
 - The goal is to reverse the symptoms of neural dysfunction.
 - Left untreated, **CES can result in permanent paralysis and incontinence.**
 - Those experiencing any of the red flag symptoms should consult a neurosurgeon as soon as possible.
 - Prompt surgery is the best treatment for patients with CES. Treating patients within 48 hours after the onset of the syndrome provides a significant advantage in improving sensory and motor deficits as well as urinary and rectal function.
 - But even patients who undergo surgery after the 48-hour ideal timeframe may experience considerable improvement.
 - Although short-term recovery of bladder function may lag behind reversal of lower extremity motor deficits, the function may continue to improve years after surgery.
 - Following surgery, drug therapy coupled with intermittent self-catheterization can help lead to slow, but steady recovery of bladder and bowel function.

III. SPINAL INFECTIONS

OVERVIEW

- Spinal infections can be classified by the anatomical location involved: the vertebral column, intervertebral disc space, the spinal canal, and adjacent soft tissues.
- Infection may be caused by bacteria or fungal organisms, and can occur after surgery.
- Most postoperative infections occur between **three days and three months** post surgery.
 - **Vertebral Infection:**
 - **Vertebral osteomyelitis** is the most common form.
 - It can develop from *direct open spinal trauma, infections in surrounding areas, and from bacteria that spreads to a vertebra.*
 - **Intervertebral Disc Space Infections:** involve the space between adjacent vertebrae. Disc space infections can be divided into three subcategories:
 - *Adult hematogenous (spontaneous),*
 - *Childhood (discitis), and*
 - *Postoperative.*
 - **Spinal canal infections:**
 - **Spinal epidural abscess:** infection that develops in the space around the dura.
 - **Subdural abscess:** is far rarer and affects the potential space between the dura and arachnoid.
 - **Intramedullary abscesses:** Infections within the spinal cord parenchyma (primary tissue)
 - **Adjacent soft-tissue infections:**
 - Include **cervical and thoracic paraspinal lesions** and **lumbar psoas muscle abscesses.**
 - Soft-tissue infections generally affect younger patients and are not seen often in older people.
- **RISK FACTORS FOR DEVELOPING SPINAL INFECTION**
 - *Advanced age*
 - *Intravenous drug use (IVDU)*
 - *HIV infection*
 - *Long-term systemic usage of steroids*
 - *Diabetes mellitus*
 - *Organ transplantation*
 - *Malnutrition*
 - *Cancer*
 - **SURGICAL RISK FACTORS** include:
 - Operation of long duration,
 - High blood loss,
 - Use of instrumentation,
 - Multiple or revision surgeries at the same site.
- **CAUSES**
 - Bacterial or a fungal infection: in another part of the body that has been carried into the spine through the bloodstream. The most common source of spinal infections is a bacterium called *Staphylococcus aureus*, followed by *Escherichia coli*.

- **Urological procedure:** veins in the lower spine come up through the pelvis, the most common area of the spine affected is the **lumbar region**.
- **Intravenous drug abusers** are more prone to infections affecting the **cervical region**.

• SYMPTOMS

- Symptoms vary depending on the type of spinal infection, but generally, pain is localized initially at the site of the infection.
- In postoperative patients, these additional symptoms may be present:
 - Wound drainage
 - Redness, swelling or tenderness near the incision

1. VERTEBRAL OSTEOMYELITIS

- Severe back pain
- Fever
- Chills
- Weight loss
- Muscle spasms
- Painful or difficult urination
- Neurological deficits

2. INTERVERTEBRAL DISC SPACE INFECTIONS

- Patients may initially have very few symptoms, but eventually develop severe back pain.
- Generally, younger, preverbal children do not have a fever nor seem to be in pain, but they **will refuse to flex their spines**. Children ages **3 to 9 typically present with back pain as the predominant symptom**.
- Postoperative disc space infection may be present after surgery, occurring on average, one month after surgery. **The pain is usually alleviated by bed rest and immobilization**, but **increases with movement**. If left untreated, the pain gets progressively worse and intractable, unresponsive even to prescription painkillers.

3. SPINAL CANAL INFECTIONS

- **Adult patients** often progress through the following clinical stages:
 - *Severe back pain with fever and local tenderness in the spinal column*
 - *Nerve root pain radiating from the infected area*
 - *Weakness of voluntary muscles and bowel/bladder dysfunction*
 - *Paralysis*
- **In children**, the most overt symptoms are prolonged crying, obvious pain when the area is palpated, and hip tenderness.

4. ADJACENT SOFT-TISSUE INFECTIONS

- In general, symptoms are usually nonspecific.
- If a **paraspinal abscess** is present, the patient may experience flank pain, abdominal pain, or a limp.
- If a **psoas muscle abscess** is present, the patient may feel pain radiating to the hip of thigh area.

DIAGNOSIS

- The biggest challenge is making an early diagnosis before serious morbidity occurs.
- Diagnosis typically takes an average of one month, but can take as long as six months, impeding effective and timely treatment. Many patients do not seek medical attention until their symptoms become severe or debilitating.

LABORATORY TESTS

- **Blood tests:** ESR, CRP, FBC, Blood cultures.
- **CT-guided biopsy** sampling of the vertebra or disc space.

IMAGING TOOLS

- **Vertebral osteomyelitis:** **CT** (The degree of bone destruction) and **MRI** (soft tissue involvement).
- **Intervertebral disc space infections:** If childhood discitis is suspected, **Plain X-Rays** are ordered first, followed by **MRI** if the x-rays are negative. For postoperative disc space infection, **MRI** is the imaging tool of choice.
- **Spinal canal infections:** **MRI with Gd enhancement (MRI contrast media)** has become the gold standard, largely replacing myelography to provide high resolution images of neural structures. If the MRI comes back negative and infection is still suspected, imaging should be repeated.
- **Adjacent soft-tissue infections:** Both **MRI and CT scan** can accurately identify soft-tissue abscesses.

NONSURGICAL TREATMENT

- Spinal infections often require **long-term intravenous antibiotic or antifungal therapy** and can equate to extended hospitalization time for the patient.
- Immobilization may be recommended when there is significant pain or the potential for spine instability.
- If the patient is neurologically and structurally stable, antibiotic treatment should be administered after the organism causing the infection is properly identified.
- Patients generally undergo antimicrobial therapy for a **minimum of six to eight weeks**.
- The type of medication is determined on a case-by-case basis depending on the patient's specific circumstances, including his or her age.

SURGICAL TREATMENT

- Nonsurgical treatment should be considered first when patients have minimal or no neurological deficits and the morbidity and mortality rate of surgical intervention is high.
- However, surgery may be indicated when any of the following situations are present:
 - *Significant bone involvement*
 - *Neurological deficits*
 - *Sepsis with clinical toxicity caused by an abscess unresponsive to antibiotics*
 - *Failure of needle biopsy to obtain needed cultures*
 - *Failure of intravenous antibiotics alone to eradicate the infection*

CHAPTER 8. AGGRESSIVE/DISTURBED BEHAVIOUR

• MEDICAL CAUSES OF VIOLENCE AND AGGRESSION IN PATIENTS

- Head injury
- Substance abuse and intoxication
- Underlying mental illness
- Hypoxia
- Metabolic disturbances/ Hypoglycaemia
- Infection: meningitis, encephalitis, sepsis
- Hyperthermia or hypothermia
- Seizures: post ictal or status epilepticus
- Vascular: stroke or subarachnoid haemorrhage

• RISK FACTORS FOR SUDDEN RELATED VIOLENCE

- Younger age
- Male gender
- History of physical abuse by parent or guardian
- History of violence
- Past juvenile detention
- Victimization in past year
- Lower income
- Unemployed and looking for work in the past
- Substance dependence only
- Comorbid mental health and substance disorder

• INVESTIGATING THE VIOLENT AND AGGRESSIVE PATIENT

- Blood sugar level
- Full blood count
- Urea, Electrolytes, Creatinine
- Paracetamol, Ethanol level
- Urinalysis
- Urine drug screen if available
- +/- Head CT/MRI
- +/- Lumbar Puncture

ED MANAGEMENT FOR VIOLENCE AND AGGRESSION

1. DE-ESCALATION STRATEGIES

- Consider personal safety at all times
- Consider the safety of other patients and their visitors at all times
- Place the person in a quiet and secure area and let staff know what is happening and why
- Never turn your back on the individual
- Don't walk ahead of the individual and ensure adequate personal space
- Provide continuous observation and record behaviour changes in patient notes
- Wear personal duress alarm if available
- Let the person talk (everyone has a story to tell, let them tell it)
- Never block off exits and ensure you have a safe escape route

2. INDICATIONS FOR RESTRAINING AND SEDATING A VIOLENT PATIENT

- Preventing harm to the patient
- Preventing harm to other patients
- Preventing harm to caregivers and other staff
- Preventing serious disruption or damage to the environment
- To assist in assessing and management off the patient
- Restraints should never be use for ease of convenience

3. PHYSICAL/MECHANICAL RESTRAINTS

- Clinicians should beware of local policies, laws and acts before restraining patients
- Applying physical restraint's is a team sport, **1 for each limb and 1 to lead the restraint and manage the airway (Minimum of 5 persons).**
- Physical restraint should always be followed up with chemical and mechanical restraints.
- Physical restraints need to be secure enough to restrain the patient, but able to be easily removed if the patient begins to vomit, seizure, or loose's control of their airway. *Restraints must be applied in the least restrictive maner and for the shortest period of time.*
- **Padding** should be applied between restraints and the patients to prevent neurovascular injury, and regular neurovascular observations should be perform every **15-30mins** whilst patient is physically restrained.
- The clinician ordering the restraints should **document the reason for restraints**, what limbs are restrained, how frequent neurovascular observations are needed, and when the restraints need reviewed, generally every 2 hours' restraints should be reviewed by treating clinician.

4. CHEMICAL RESTRAINTS/SEDATION

1. Benzodiazepines:

- **Midazolam** 2.5-5mg IV or IM increments and work upwards
- **Diazepam** 5-10 PO or IV increments and work upward
- **Lorazepam** 1-2mg PO

2. Antipsychotics:

- **Haloperidol** 2.5-10mg IV or IM
- **Olanzapine** 5-10mg PO or SL, or 10mg IM
- **Droperidol** 2.5-10MG IV or IM
- **Quetiapine** 25-200MG PO
- **Risperidone** 0.25-2mg PO/SL
- **Chlorpromazine** 100-200mg IV infusion over 24 hours

3. Barbiturates:

- **Thiopentone** 25mg IV increments until sedation has been achieved

Procyclidine is an anticholinergic drug principally used for the treatment of drug-induced Parkinsonism, Akathisia and Acute Dystonia; Parkinson disease; and idiopathic or secondary Dystonia.

FRCEM INTERMEDIATE EXAM QUESTION MARCH 2017

• COMPLICATIONS OF SEDATION AND RESTRAINING PATIENTS

- Respiratory Depression and Pulmonary Aspiration
- Sudden cardiac death/ Excited delirium, Hypotension/ DVT & PE
- Rhabdomyolysis/ Dystonic reactions and Neuroleptic Malignant Syndrome
- Anticholinergic effects/ Delirium/ Lactic acidosis /Lowered seizure threshold

CHAPTER 9. BLACKOUT/COLLAPSE

DIFFERENTIAL DIAGNOSIS

- Transient loss of consciousness is usually due to **syncope**.
- Other possible causes are:
 - *Hypoglycaemia*
 - *Falls/Trauma*.
 - *Epilepsy*.
 - *TIA/Stroke*.
 - *Dizziness or Vertigo without loss of consciousness*.
 - *Alcohol/Drug abuse*.
 - *Narcolepsy, Cataplexy/ Drop attacks*.
 - *Psychogenic pseudosyncope*.

I. SYNCOPE

- It is a transient loss of consciousness caused by transient global cerebral hypoperfusion characterised by rapid onset, short duration and spontaneous complete recovery.
- The term syncope excludes seizures, coma, shock or other states of altered consciousness.
- Patients presenting with a history of blackouts, faints or collapse need careful evaluation to assess the precise nature of the problem. This is essential so as to assess both the risk of a serious underlying disorder and also the risk of recurrence and subsequent injury.

CAUSES OF SYNCOPE

- **Neurally Mediated Syncope (NMS)** - also called reflex syncope:
 - **Vasovagal syncope (common faint):**
 - **Emotional:** fear, severe pain, blood phobia, unexpected sight, sound or smell.
 - **Orthostatic stress** - eg, prolonged standing or when in crowded, hot places.
 - **Situational syncope** - eg, cough, sneeze, gastrointestinal stimulation (swallowing, defecation, visceral pain), micturition.
 - **Carotid sinus hypersensitivity:** occurs when rotating the head - eg, while shaving, especially if a collar is tight or in the presence of a neck tumour.
 - **Glossopharyngeal neuralgia.**
- **Orthostatic Hypotension (postural hypotension)** - syncope occurs after standing up:
 - **Autonomic failure:**
 - Multiple system atrophy, Parkinson's disease, diabetic neuropathy, amyloidosis.
 - Medications - eg, antihypertensives.
 - Post-exercise.
 - Postprandial.
 - **Hypovolaemia:**
 - Haemorrhage.
 - Vomiting, diarrhoea and other causes of dehydration.
 - Addison's disease.
- **Cardiac Arrhythmias:**
 - Sick sinus syndrome, atrioventricular (AV) conduction system disease.
 - Paroxysmal supraventricular tachycardia, ventricular tachycardia.
 - Inherited syndromes - eg, long QT syndrome, Brugada's syndrome.
 - Malfunction of a pacemaker or implantable cardioverter defibrillator (ICD).
 - Drug-induced arrhythmias.

- **Structural cardiac or cardiopulmonary disease:**
 - Obstructive Cardiac Valvular Disease.
 - Acute Coronary Syndrome (ACS)
 - Hypertrophic obstructive cardiomyopathy (HOCM).
 - Atrial Myxoma/ Acute Aortic Dissection.
 - Pericardial Disease or Tamponade.
 - Pulmonary Embolus or Pulmonary Hypertension.
- **Cerebrovascular:**
 - Vascular Steal Syndromes - eg, subclavian steal syndrome.
- Substance abuse, alcohol intoxication.
- **Psychogenic:** factitious, anxiety, panic attacks, hyperventilation

CLINICAL PRESENTATION

- A thorough history and examination are essential.

History

- An accurate history, including from an eye-witness if available, is essential and often alone will lead to a correct diagnosis. Points to cover in the history include:
 - Was loss of consciousness (LOC) complete?
 - Was LOC temporary? How quickly did it come on and how long did it last?:
 - Was there any warning: light-headedness, nausea, sweating, weakness or visual disturbance?: Preceding nausea, sweating and blurred vision have been shown to be predictive of non-cardiac syncope in the elderly.
 - Did it occur during exercise or while lying down? Were there any palpitations or was there accompanying chest pain? Was there any shortness of breath?:
 - If so, this suggests a cardiac cause.
 - Dyspnoea has been shown to be predictive of cardiac syncope in the elderly.
 - Was recovery spontaneous and total? Were there any symptoms following recovery?:
 - Recovery from syncope is usually associated with almost immediate restoration of appropriate behaviour and orientation but there may be marked fatigue.
 - Was there loss of postural tone?
 - Was there a situational trigger?
 - Has there been a recent change in medication?:
 - New medication or a change of dose causing orthostatic hypotension.
 - Syncope may be more likely to occur in the morning.
 - Is there any family history of sudden death?

Examination

The following physical findings may indicate a likely underlying cause:

- **Syncope caused by orthostatic hypotension:**
 - There may be examination evidence of a drop-in blood pressure (usually $>20/10$ mm Hg) within three minutes of standing, associated with syncope or presyncope.
- **Cardiac syncope:** Full CVS examination may reveal a severe structural abnormality.
- **Cerebrovascular syncope:**
 - With arm exercise.
 - Differences in blood pressure or pulse in the two arms.

NB: Prolonged unconsciousness, witnessed abnormal behaviour before, during or after the event, confusion after the event, tongue biting, head turning or prolonged limb jerking, unusual posturing - all suggest a **NON-SYNCOPEAL EVENT** and should prompt referral to a specialist in epilepsy to be seen within two weeks.

SEIZURE VERSUS SYNCOPÉ

CLINICAL FEATURES OF SYNCOPÉ		
Feature	Seizure	Syncopé
Trigger	Rare	Common
Prodrome	Aura – unpleasant smell, epigastric sensation	Presyncopal features like nausea, sweating, pallor
Onset	Sudden	Gradual
Duration	1–3 minutes	1–30 seconds
Colour	Cyanosed	Usually pale
Convulsions	Tonic-clonic movements, automatism, neck turned to one side	May have movement after loss of consciousness
Tongue bite	Common, on the side	Rare, usually on the tip
Post event	Confusion, aching muscles, joint dislocations	Rapid recovery, nausea or vomiting afterwards

INVESTIGATIONS AND ASSESSMENT

Investigations are guided by the history and examination. Initial tests in primary care include:

- **Orthostatic blood pressure** measurement.
- **ECG:** there may be evidence of ischaemia or arrhythmias.
- **FBC** if anaemia or bleeding is suspected (acute anaemia will cause syncope but patients adapt in cases of chronic anaemia).
- **Fasting blood glucose**, if hypoglycaemia is a possibility.
- In most cases, the initial assessment will lead to a definite, or at least a likely, diagnosis, which will clarify the selection of further investigations and management.
- However, syncope is often multifactorial, especially in older individuals.

RISK STRATIFICATION

- It is essential to assess the risk of major cardiovascular events or sudden cardiac death.
- There are several risk scores to help identify those patients with syncope who are at high risk of adverse events but none of the scores is widely accepted:
 - **Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) score.**
 - **San Francisco Syncope Rule (SFSR):** this is the simplest, and uses an abnormal ECG, heart failure, anaemia and systolic hypotension (below 90 mm Hg) to identify patients who require urgent action.
 - **European Guidelines in Syncope Study (EGSYS) score.**

The San Francisco Syncope Rule: CHES

Congestive cardiac failure history

Haematocrit < 30%

ECG abnormality; new, any non-sinus rhythm

Shortness of breath

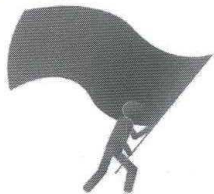
Systolic Blood Pressure <90 mm Hg

The presence of any factor is considered sufficient for the patient to be high risk.

UNCOMPLICATED SYNCOPE (VASOVAGAL): 3 Ps

- **Posture:** Prolonged standing, similar episodes prevented by lying down
- **Provoking:** Pain, procedures
- **Prodromes:** Sweating, Feeling Warm / Hot Prior To TLOC

RED FLAGS



- Abnormal ECG
- History of heart failure
- Syncope during exertion
- Family hx of sudden death in people aged younger than 40 years and/or inherited cardiac condition
- New or unexplained breathlessness
- A heart murmur

- Consider referring within 24 hours for cardiovascular assessment, as above, anyone aged 65+ who has experienced TLOC without prodromal symptoms

ED MANAGEMENT OF TLOC

- History
- Examination
- Excluded other differential diagnosis and manage accordingly.
- **If syncope:**
 - ECG: abnormal>>> refer; Normal>>> Proceed below
 - Red flags: Refer
 - Exclude the 3 P's of low risk
 - Situational
 - Orthostatic
 - Unexplained: non-urgent CV assessment
- **Seek and treat underlying cause and complications**
 - If no obvious cause found, the main management issue in ED is to determine appropriate disposition.
- **LOWER THRESHOLD FOR ADMISSION IF:**
 - Syncope unwitnessed
 - Significant risk factors, including:
 - Cardiovascular disease
 - Documented or suspected arrhythmias
 - Known epileptic with greater than one seizure or without home supervision
 - Cardiac pacemaker or other devices
 - Elderly
 - Suspected cardiac cause:
 - Admit for monitoring and cardiology review (do not sent home for a Holter monitor)
 - Need to rule out an ischemic event and / or arrhythmias
- **HIGH RISK FACTORS FOR A CARDIAC CAUSE INCLUDE:**
 - Age
 - Known electrophysiological abnormalities, or previously documented malignant arrhythmias

- Diabetes
- Newly abnormal ECG
- Elevated troponin level
- Significant depression of ventricular function, documented on echocardiogram
- Documented IHD including past STEMI, non-STEMI, abnormal cardiac functional study or abnormal angiogram
- Patients with pacemakers or other cardiac devices:
 - Have a high index of suspicion in these patients for arrhythmia and / or cardiac device malfunction
 - All patients with pacemakers with unexplained collapse must be admitted until such time as their pacemaker can be checked
 - Most devices can be interrogated for a record of significant arrhythmia over an extended period of weeks
- **Suspected drug related cause**
 - These patients should be admitted for drug medication review and observation
- **Vasovagal**
 - Even if the cause is "benign", consider admission should still be considered in elderly patients or those with significant co-morbidities.
 - **This is particularly important when:**
 - Episodes have been recurrent
 - Significant injuries have occurred
 - Lack of supervision at home
- **A short stay admission may be appropriate for:**
 - Observation
 - Care coordination
 - Aged Care Assessment Team (ACAT) assessment
 - physiotherapy assessment

DISCHARGE CRITERIA

- **Patients can usually be discharged if:**
 - They do not have significant clinical risk factors, including (**3Ps with No Red Flags**):
 - Abnormal ECG
 - CVS risk factors
 - Initial hypotension
 - Initial history of shortness of breath
 - Witnessed seizure activity or a history of seizures, especially when the event is unwitnessed
 - Observations and clinical findings are normal
 - Medications reviewed
 - Safe home environment (especially the elderly)
- **If uncertain then observe for 24 hours**

II. DRIVING AND COMMON ED CONDITIONS

- In the UK, following a single vasovagal syncope, driving is not restricted and the Driver and Vehicle Licensing Agency (DVLA) does not need to be informed.
- If recurrent, on each occasion it must be due to strong Provocation, associated with Prodromal symptoms and Posture, ie it is unlikely to occur while sitting or lying - the '3 Ps'. Greater restrictions apply if the situation is more complicated, such as cough syncope, or if diagnosis is less clear. If in doubt, contact the DVLA.

DVLA STANDARDS OF FITNESS TO DRIVE OF COMMON ED CONDITIONS

DISORDER	CAR OR MOTORCYCLE	BUS OR LORRY
REFLEX VASOVAGAL SYNCOPE: Syncope with the "3Ps" (Provocation/ Prodrome/ Postural) If recurrent, will need to check the "3 Ps" apply on each occasion.	No driving restrictions. (Except Cough Syncope) DVLA need not be notified.	No driving restrictions (Except Cough Syncope) DVLA need not be notified
LOSS OF CONSCIOUSNESS/ LOSS OF OR ALTERED AWARENESS: likely to be unexplained syncope but with a high probability of reflex vasovagal syncope	No driving restrictions. DVLA need not be notified.	Can drive 3 months after the event. (Except Cough Syncope)
LOSS OF CONSCIOUSNESS/ LOSS OF OR ALTERED AWARENESS with High Risk Factors. (Includes > 1 episode in previous 6 months)	Licence refused/revoked for 6 months if no cause identified. Can drive 4 weeks after the event if the cause has been identified and treated.	Licence refused/revoked for 12 months if no cause identified. Can drive 3 months after the event if the cause has been identified and treated.
COUGH SYNCOPE	Driving must cease for 6 months if a single episode, increased to 12 months if multiple attacks.	5 years off driving from the date of the last attack.
FIRST UNPROVOKED EPILEPTIC SEIZURE / SOLITARY FIT	6 months off driving from the date of the seizure	5 years off driving from the date of the seizure.
PRESUMED LOSS OF CONSCIOUSNESS/loss of or altered awareness with Seizure markers.	6 months off driving from the date of episode. If a person suffers recurrent episodes of LOC with seizure markers, 12 months' freedom from such episodes must be attained.	5 years off driving from the date of an episode if the licence holder has undergone assessment by an appropriate specialist and no relevant abnormality has been identified.
CEREBROVASCULAR DISEASE: including stroke due to occlusive vascular disease, spontaneous intracerebral haemorrhage, TIA, amaurosis fugax and intracranial venous thrombosis.	Must not drive for 1 month . May resume driving after this period if the clinical recovery is satisfactory. There is no need to notify DVLA unless there is residual neurological deficit 1 month after the episode; Multiple TIAs over a short period may require at least 3 months free from further attacks before resuming driving and should notify DVLA.	Licence refused or revoked for 1 year following a stroke or TIA. Can be considered for licensing after this period provided that there is no debarring residual impairment likely to affect safe driving and there are no other significant risk factors.

DISORDER	CAR OR MOTORCYCLE	BUS OR LORRY
ANGINA	Driving must cease when symptoms occur at rest, with emotion or at the wheel. Driving may recommence when satisfactory symptom control is achieved. DVLA need not be notified.	Refusal or revocation with continuing symptoms (treated and/or untreated) Re-licensing may be permitted thereafter provided: Free from angina for at least 6/52; The exercise or other functional test requirements can be met and There is no other disqualifying condition.
ACUTE CORONARY SYNDROMES (ACS)	If successfully treated by coronary angioplasty, driving may recommence after 1 week . If not successfully treated by coronary angioplasty, driving may recommence after 4 weeks provided: • There is no other disqualifying condition. DVLA need not be notified.	All Acute Coronary Syndromes disqualify the licence holder from driving for at least 6 weeks . Re/licensing may be permitted thereafter provided: • The exercise or other functional test requirements can be met. • There is no other disqualifying condition.
ARRHYTHMIA Sinoatrial disease Significant atrio-ventricular conduction defect Atrial flutter/fibrillation Narrow or broad complex tachycardia.	Driving must cease if the arrhythmia has caused or is likely to cause incapacity. Driving may be permitted when underlying cause has been identified and controlled for at least 4 weeks . DVLA need not be notified unless there are distracting/disabling symptoms.	Disqualifies from driving if the arrhythmia has caused or is likely to cause incapacity.
HYPERTENSION	Driving may continue unless treatment causes unacceptable side effects. DVLA need not be notified.	Disqualifies from driving if resting BP consistently >180/100 mm Hg.
DIABETICS with Impaired awareness of Hypoglycaemia	If confirmed, driving must stop . Driving may resume provided reports show awareness of hypoglycaemia has been regained, confirmed by consultant/GP report.	If confirmed, driving must stop . Driving may resume provided reports show awareness of hypoglycaemia has been regained, and there are no other debarring complications of DM such as a visual field defect.
PERSISTENT ALCOHOL MISUSE	Licence revocation or refusal until a minimum 6-month period of controlled drinking or abstinence has been attained, with normalisation of blood parameters.	Revocation or refusal of a vocational licence until at least 1 year period of abstinence or controlled drinking has been attained, with normalisation of blood parameters.
ALCOHOL DEPENDENCE	Licence revocation or refusal until a 1 year period free from alcohol problems has been attained.	Vocational licensing will not be granted where there is a history of alcohol dependence within the past 3 years .
LIABILITY TO SUDDEN ATTACKS OF UNPROVOKED OR UNPRECIPITATED DISABLING GIDDINESS	Cease driving on diagnosis. Driving will be permitted when satisfactory control of symptoms achieved.	Licence refused or revoked if condition sudden and disabling. Must be symptom free and completely controlled for at least 1 year from last attack before re-application.

CHAPTER 10. BREATHLESSNESS & COUGH

I. ASTHMA

PEAK EXPIRATORY FLOW RATE

- Peak expiratory flow (PEF) measurement should be performed on all patients that are capable of performing the test. PEFR is effort dependent and the best of three attempts should be recorded. This process **should not be performed in hypoxic patients** and should be stopped if a patient **becomes hypoxic during testing**.
- Serial measurements allow response to therapy to be assessed.
- The benchmark for comparison should be the patients best PEFR in the last 2 years.
- Patients commonly cannot recall their best PEFR and this may have to be predicted from charts based on their age, sex and height.

RISK STRATIFICATION

- The British Asthma guidelines risk stratify adults with acute asthmatic exacerbations into one of the following categories:
 - *Near fatal asthma*
 - *Life-threatening asthma*
 - *Acute severe asthma*
 - *Moderate asthma exacerbation*
 - *Mild asthma exacerbation*

BTS ASTHMA SEVERITY

BTS ASTHMA ASSESSMENT			
Near Fatal Asthma	Fatal	↑PaCO ₂ and/or requiring mechanical ventilation with raised inflation pressures	
Life Threatening Asthma		Any one of the following in a patient with severe asthma: PEF <33% best or predicted SpO ₂ <92% PaO ₂ <8 kPa normal PaCO ₂ (4.6-6.0 kPa)	
		Silent chest Cyanosis Feeble resp effort Bradycardia	Dysrhythmia Hypotension Exhaustion Confusion/coma
Acute asthma	severe	Any one of: PEF 33-50% best or predicted Resp rate >25/min Heart rate >110/min	
		Inability to complete sentences in one breath	
Moderate asthma exacerbation		Increasing symptoms PEF >50-75% best or predicted	No features of acute severe asthma
Brittle asthma		Type 1: wide PEF variability (>40% diurnal variation for >50% of the time over a period >150 days) despite intense therapy Type 2: sudden severe attacks on a background of apparently well-controlled asthma	

PASS SCORE

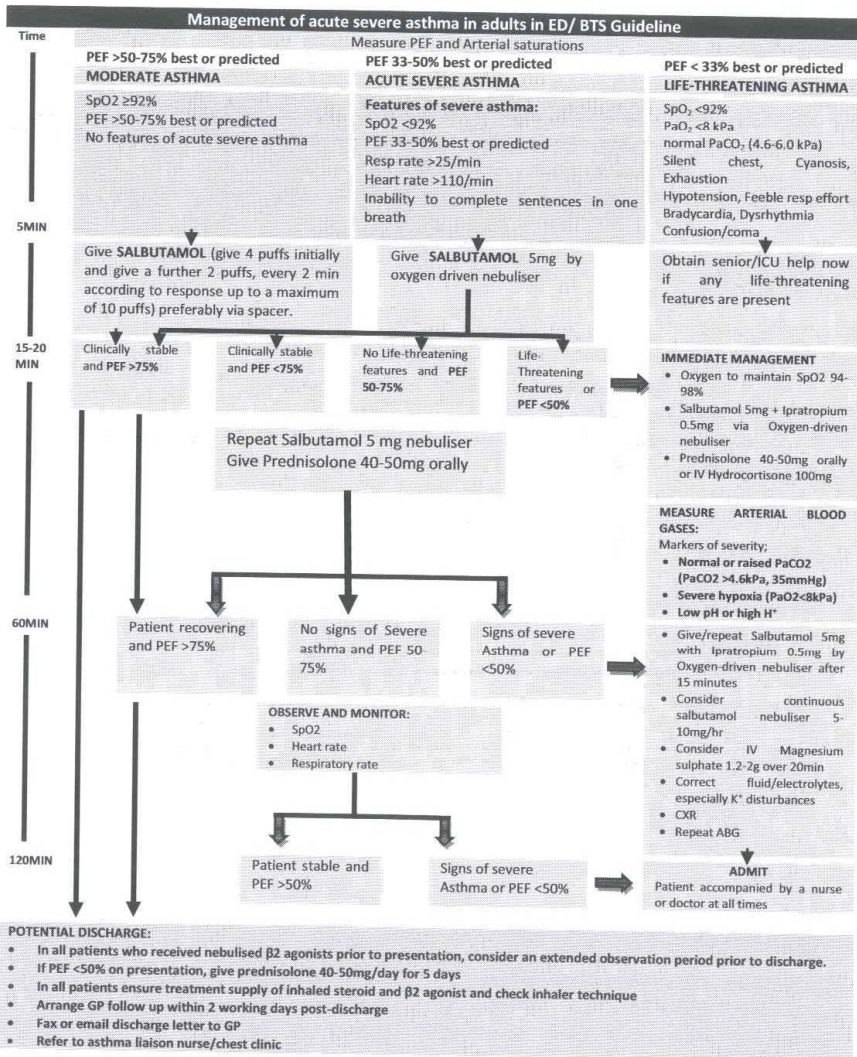
PASS (PAEDIATRIC ASTHMA SEVERITY SCORE)			
	0	1	2
Wheeze	None	Moderate	Severe/absent
Work of breathing	None	Moderate	Severe
Prolonged Expiration	None	Moderate	Severe

Score ≤4: Mild Score >4-<7: Moderate Score ≥7: Severe

1. ACUTE SEVERE ASTHMA IN ADULT

- This is characterized by any one of:
 - **Severe breathlessness:**
 - An inability to complete a sentence in one breath;
 - A silent chest;
 - Cyanosis.
 - **Tachypnoea:** respiratory rate >25 breaths/min.
 - **Tachycardia:** heart rate >110 beats/min.
 - **Peak expiratory flow (PEF) 33–50%** of best or predicted.
- Acute severe asthma is considered *life threatening* in a patient with any one of the following:
 - Feeble respiratory effort;
 - PEF <33% of best or predicted;
 - SpO₂ <92%;
 - PaO₂ <8kPa;
 - Normal PaCO₂ (4.6–6.0kPa);
 - Cyanosis;
 - Bradycardia, arrhythmias, hypotension;
 - Exhaustion, confusion, coma.
- **IMMEDIATE MANAGEMENT OF ACUTE SEVERE ASTHMA**
 - High-flow oxygen.
 - High-dose beta-2 agonists via oxygen driven nebulizer.
 - Salbutamol 5mg, terbutaline 10mg.
 - Ipratropium bromide, 0.5mg via oxygen driven nebulizer.
 - Prednisolone 40–50mg orally, or hydrocortisone 100mg IV, or both.
 - **Monitor**
 - PEF 15–30min intervals.
 - **Pulse oximetry:** maintain SpO₂ > 92%.
 - **Arterial blood gases.**
 - **A chest X-ray is only indicated if:**
 - *There is suspected pneumothorax or pneumo-mediastinum;*
 - *There is suspected consolidation;*
 - *There is failure to respond to therapy;*
 - *Mechanical ventilation is required.*
- **SUBSEQUENT MANAGEMENT**
 - **If the patient is improving:**
 - Continue oxygen therapy;
 - Give IV Hydrocortisone 100mg 6 hourly or 40–50mg orally daily;
 - Give nebulized salbutamol and Ipratropium 4–6 hourly.
 - **If the patient is not improving:**
 - Continue oxygen therapy;
 - Give nebulized salbutamol 5mg more frequently, every 15–30mins or 10mg continuously hourly;
 - Continue Ipratropium 0.5mg 4–6 hourly;
 - Give Magnesium Sulphate 1.2–2.0g IV as slow infusion over 20mins;
 - Consider IV beta-2 agonist or aminophylline;
 - Consider need for tracheal intubation and Mechanical ventilation.

- **Discuss with Critical Care team if there is:**
 - Need for tracheal intubation and ventilatory support;
 - Continuing failure to respond to treatment;
 - A deteriorating PEF;
 - Persistent or worsening hypoxia;
 - Hypercapnia;
 - Development of acidosis (fall in pH or increase in hydrogen ion concentration);
 - Exhaustion; Drowsiness or confusion; Coma
 - Respiratory arrest.



2. MANAGEMENT ASTHMA IN LESS THAN 2-YEAR-OLD

- The BTS guidelines on acute asthma in children recommend that **oral steroids** should be given early in the treatment of acute asthma attacks.
- The following is advised:
 - Use a dose of:
 - **Children < 2 years: 10mg Prednisolone**
 - **Children aged 2–5 years: 20 mg Prednisolone**
 - **Children >5 years: 30–40 mg Prednisolone**
 - Those already receiving maintenance steroid tablets should receive **2 mg/kg prednisolone up to a maximum dose of 60 mg.**
 - **Repeat the dose** of prednisolone in children who vomit and consider intravenous steroids in those who are unable to retain orally ingested medication.
 - Treatment for **up to three days** is usually sufficient, but the length of course should be tailored to the number of days necessary to bring about recovery.
 - Tapering is unnecessary unless the course of steroids exceeds 14 days.

MANAGEMENT OF ACUTE ASTHMA IN CHILDREN AGED UNDER 2 YEARS

- The assessment of acute asthma in early childhood can be difficult
- Intermittent wheezing attacks are usually due to viral infection and the response to asthma medication is inconsistent
- Prematurity and low birth weight are risk factors for recurrent wheezing
- The differential diagnosis of symptoms includes:
 - Aspiration pneumonitis
 - Pneumonia
 - Bronchiolitis
 - Tracheomalacia
 - Complications of underlying conditions such as congenital anomalies and cystic fibrosis

TREATMENT OF ACUTE ASTHMA**BRONCHODILATORS**

- | | |
|----------|---|
| B | Oral β_2 agonists are not recommended for acute asthma in infants. |
| A | For mild to moderate acute asthma attacks, a pMDI + spacer and mask is the optimal drug delivery device. |
| B | Consider inhaled ipratropium bromide in combination with an inhaled β_2 agonist for more severe symptoms. |

STEROID THERAPY

- | | |
|----------|---|
| B | In infants, consider steroid tablets early in the management of severe asthma attacks in the hospital setting. |
| ✓ | Steroid tablet therapy (10 mg of soluble prednisolone for up to three days) is the preferred steroid preparation for use in this age group. |

For children with frequent episodes of wheeze associated with viruses caution should be taken in prescribing multiple courses of oral steroids.

3. MANAGEMENT ASTHMA BETWEEN 2 – 5-YEAR-OLD

- The below flow diagram was copied from the joint BTS/SIGN guidelines 2011

ASSESS ASTHMA SEVERITY

MODERATE ASTHMA

- SpO₂ ≥92%
- No features of acute severe asthma

NB: If a patient has signs and symptoms across categories, always treat according to their most severe features

SEVERE ASTHMA

- SpO₂ <92%
- Too Breathless to talk
- Resp rate >40/min
- Heart rate >140/min
- Use of accessory neck muscles

LIFE-THREATENING ASTHMA

- SpO₂ <92% plus any of:
 - Silent chest,
 - Poor resp effort
 - Agitation
 - Cyanosis
 - Altered consciousness

Oxygen via face mask/ nasal prongs to achieve SpO₂ 94-98%

- β₂ agonist** 2-10 puffs via spacer ± facemask (given one at a time single puffs, tidal breathing and inhaled separately)
- Increase **β₂ agonist** dose by 2 puffs every 2 minutes up to 10 puffs according to response
- consider soluble oral **Prednisolone** 20mg

- β₂ agonist** 10 puffs via spacer ± facemask or nebulised **Salbutamol** 2.5mg or **Terbutaline** 5mg.
- Soluble **Prednisolone** 20mg or IV **Hydrocortisone** 4mg/kg
- Repeat β₂ agonist up to every 20-30min according to response
- if poor response, add 0.25mg nebulised **Ipratropium bromide**

- Nebulised **β₂ agonist**:
 - Salbutamol** 2.5 mg or **Terbutaline** 5mg + **Ipratropium bromide** 0.25mg nebulised.
- Oral **Prednisolone** 20mg or IV **Hydrocortisone** 4mg/kg if vomiting.
- Discuss with senior clinician, PICU team or paediatrician.
- Repeat bronchodilators every 20-30minutes

Reassess within 1 hour

ASSESS RESPONSE TO TREATMENT

Record RR, HR and Oxygen saturation every 1-4hours

RESPONDING

- Continue bronchodilators 1-4hours prn
- Discharge when stable on 4 hourly treatment
- Continue oral Prednisolone for up to 3 days

At Discharge

- Unsure stable on 4 hly inhaled treatment
- Review the need for regular treatment and the use of inhaled steroids
- Review inhaler technique
- Provide a written asthma action plan for treating future attacks
- Arrange follow up according to local policy

NOT RESPONDING

- Arrange HDU/PICU transfer

Consider

- CXR and Blood gases**
- IV Salbutamol** 15mcg/Kg bolus over 10min followed by continuous infusion 1-5mcg/kg/min (dilute to 200mcg/ml)
- IV Aminophylline** 5mg/Kg loading dose over 20min (omit in those receiving oral theophyllines) followed by continuous infusion 1mg/kg/hour.

4. MANAGEMENT ASTHMA >5-YEAR-OLD

- The below flow diagram was copied from the joint BTS/SIGN guidelines 2011

ASSESS ASTHMA SEVERITY**MODERATE ASTHMA**

- SpO₂ ≥92%
- PEF >50% Best or predicted
- No features of acute severe asthma

NB: If a patient has signs and symptoms across categories, always treat according to their most severe features

SEVERE ASTHMA

- SpO₂ <92%
- PEF 33-50% best or predicted
- Resp rate >30/min
- Heart rate >125/min
- Use of accessory neck muscles

LIFE-THREATENING ASTHMA

SpO₂ <92% plus any of:

- PEF < 33% best or predicted
- Silent chest,
- Poor resp effort
- Cyanosis
- Altered consciousness

Oxygen via face mask/ nasal prongs to achieve SpO₂ 94-98%

- β₂ agonist 2-10 puffs via spacer
- Increase β₂ agonist dose by 2 puffs every 2 minutes up to 10 puffs according to response
- Oral Prednisolone 30-40 mg

- β₂ agonist 10 puffs via spacer or nebulised Salbutamol 2.5 - 5mg or Terbutaline 5 -10mg.
- Oral Prednisolone 30-40mg or IV Hydrocortisone 4mg/kg
- Repeat β₂ agonist up to every 20-30min according to response
- if poor response, add 0.25mg nebulised Ipratropium bromide

- Nebulised β₂ agonist:
- Salbutamol 5 mg or Terbutaline 10mg + Ipratropium bromide 0.25 mg nebulised.
- Oral Prednisolone 30-40mg or IV Hydrocortisone 4mg/kg if vomiting.
- Discuss with senior clinician, PICU team or paediatrician.
- Repeat bronchodilators every 20-30minutes

Reassess within 1 hour

ASSESS RESPONSE TO TREATMENT

Record RR, HR, Oxygen saturation and PEF/FEV every 1-4hours

RESPONDING

- Continue bronchodilators 1-4hours prn
- Discharge when stable on 4 hourly treatment
- Continue oral Prednisolone for up to 3 days

At Discharge

- Unsure stable on 4 hly inhaled treatment
- Review the need for regular treatment and the use of inhaled steroids
- Review inhaler technique
- Provide a written asthma action plan for treating future attacks
- Arrange follow up according to local policy

NOT RESPONDING

- Arrange HDU/PICU transfer

Consider

- CXR and Blood gases
- IV Salbutamol 15mcg/Kg bolus over 10min followed by continuous infusion 1-5mcg/kg/min (dilute to 200mcg/ml)
- IV Aminophylline 5mg/Kg loading dose over 20min (omit in those receiving oral theophyllines) followed by continuous infusion 1mg/kg/hour.

II. COPD

INTRODUCTION

- COPD is a respiratory disease characterised by airflow obstruction that is not fully reversible. Airflow obstruction is defined as a value of <0.7 when a ratio of the FEV₁ (Forced expiratory volume in 1 second) / FVC (Forced vital capacity) is measured.
- A diagnosis of COPD should be considered in a patient over the age of **35** who presents with exertional breathlessness, cough, sputum production, wheeze or frequent winter bronchitis **in the presence of risk factors**.
- Traditionally within the diagnosis of COPD there were considered to be two main subtypes, namely **chronic bronchitis and emphysema**. COPD is now the preferred term for all with the disease.
- **An exacerbation of COPD** is defined as a worsening of the patient's symptoms beyond their normal day-to-day variability.
- Additional medication will usually be needed in order to treat the exacerbation.
- **The cardinal symptoms of COPD are:**
 - Exertional breathlessness
 - Cough
 - Sputum production
 - Wheeze
 - Frequent winter bronchitis
 - Usually these will occur in a person over 35 years old and with risk factors.
- **RISK FACTORS**
 - **Smoking** is by far the largest risk factor for COPD
 - Occupational exposure to fumes or dust
 - Occupational exposure to tobacco smoke
 - Alpha 1 antitrypsin deficiency.

AETIOLOGY OF EXACERBATIONS

- Most COPD exacerbations are due to viral or bacterial infections of the respiratory tract, however in some cases they are caused by environmental pollution.
- Up to 30% have an unknown aetiology.

DIAGNOSIS OF COPD

- **Clinical factors that may help differentiate asthma and COPD**

FEATURE	COPD	ASTHMA
Smoker or ex-smoker	Nearly all	Possible
Symptoms aged < 35 years	Rare	Often
Chronic productive cough	Common	Uncommon
Breathlessness	Persistent and progressive	Variable
Night waking with SOB/Wheeze	Uncommon	Common
Diurnal or day to day variability of symptoms	Uncommon	Common

Nice 2010 Guidelines

INVESTIGATIONS

- Investigations that should be performed in the ED when a patient is presenting with an exacerbation of COPD include:
 - Arterial blood gas analysis:** to evaluate evidence of acidosis, hypercapnea and hypoxaemia
 - CXR:** to look for evidence of consolidation, exclude pneumothorax and exclude other pathologies which may cause increased breathlessness
 - ECG:** to exclude other or concurrent causes of breathlessness such as ischaemic heart disease or signs of pulmonary embolism. In severe disease there may be signs of pulmonary hypertension such as peaked p waves or right ventricular hypertrophy
 - Full blood count:** This may identify anaemia as a cause of breathlessness or show evidence of secondary polycythaemia.
 - Urea and electrolytes**
 - Theophylline level** if the patient is already on theophylline therapy
 - Sputum analysis:** if sputum is purulent a sample should be sent for microscopy, culture and sensitivity
 - Blood cultures** if pyrexia present

CLASSIFICATION OF COPD

PD CLASSIFICATION (POST BRONCHODILATORS)			
		Post bronchodilator FEV1/FVC	FEV1 % Predicted
Stage 1	Mild	<0.7	> 80%
Stage 2	Moderate	<0.7	50-79%
Stage 3	Severe	<0.7	30-49%
Stage 4	Very severe	<0.7	<30%

ED MANAGEMENT OF COPD

- Bronchodilators and oxygen therapy:**
 - The most commonly used bronchodilators in the ED are **Beta 2 agonists** such as **salbutamol** and **terbutaline**, and anticholinergics such as **ipratropium bromide**
 - If a patient is **acidotic or hypercapnic** nebulisers should be **driven by air not oxygen***
 - Oxygen should be given to maintain saturations in a targeted range which should normally be **88-92%**.
- Steroids:**
 - Oral corticosteroids should be used in all patients admitted to hospital
 - Prednisolone 30mg for 7 to 14 days**
- Antibiotics:**
 - Antibiotics should be given to those with purulent sputum or those with clinical signs of pneumonia or CXR changes.
 - Empirical antibiotic therapy should be with **aminopenicillin, macrolide or tetracycline** unless local microbiological policy states otherwise
- Theophylline / Aminophylline:**
 - Intravenous aminophylline should be considered **only if there is an inadequate response to nebulised bronchodilators.**
 - The loading dose of aminophylline should be omitted in patients taking oral theophylline*

- o *The dose of oral theophylline should be reduced at the time of an exacerbation if the patient needs concurrent macrolide or fluoroquinolone antibiotics*
- **NON-INVASIVE VENTILATION:**
 - o Non-invasive ventilation should be used as the treatment of choice for **hypercapnic respiratory failure** if optimal medical therapy has not been successful.
 - o **Optimal Medical Therapy:** The Royal College of Physicians guideline states that maximum medical treatment includes:
 - Controlled **oxygen** therapy to maintain **SaO₂ 88-92%**
 - **Nebulised salbutamol** 2.5-5 mg
 - **Nebulised Ipratropium** 500 micrograms
 - **Prednisolone** 30 mg
 - **Antibiotic** agent when indicated
 - o **NIV** should be considered **within 60 minutes** of arrival to hospital in all patients with an exacerbation of COPD and a persistent respiratory acidosis in whom the above treatment has been unsuccessful.
 - o Non-invasive ventilation used as an adjunct to standard care has been found to be associated with lower mortality, lower need for intubation, lower likelihood of treatment failure and shorter duration of stay in hospital.
- **Other therapy:**
 - o **Hospital at home** and **assisted discharge schemes** are safe, effective and should be considered in patients who would otherwise require hospital admission.
 - o **Smoking cessation**

PROGNOSIS

- A UK audit has shown death in 14% of patients admitted to hospital within 3 months of admission.
- **The most important prognosticators for death in this group were:**
 - o Poor performance status*
 - o Low arterial pH on admission*
 - o Presence of bilateral leg oedema*
 - o Age >70
 - o Home circumstances, particularly if the patient is in a nursing home
 - o Unrecordable peak flow on admission
 - o Pulse oximetry showing oxygen saturation under 86%
 - o Intervention with assisted ventilation
- The 3 marked with * were the 3 major independent predictors of mortality.

III. PNEUMONIA

AETIOLOGY

- A single pathogen is identified in 85% of patients where an aetiology is found, however the true frequency of polymicrobial Community Acquired Pneumonia (CAP) is not clear.
 - **Streptococcus pneumoniae** is the most frequently identified pathogen (39%).
 - **Chlamydophila pneumoniae** is identified in 13% of hospitalised patients but the incidence in community cases is unknown.
 - **Mycoplasma pneumoniae** occurs in epidemics spanning three winters every four years and therefore its incidence is variable.
 - Although **legionella species** and **staphylococcus aureus** are identified more frequently in patients managed on the ICU, **streptococcus pneumoniae** is still the most frequent isolated organism in this setting.
 - Gram-negative enteric organisms, **chlamydophila psittaci** and **coxiella burnetii** are uncommon causes of CAP.
 - Legionella and mycoplasma species are less commonly isolated in elderly patients with CAP but otherwise the causative organisms have a similar frequency as those found in younger patients.
- **Atypical pneumonia**
 - The BTS considers the term atypical pneumonia misleading, as it incorrectly implies a distinct clinical pattern. Instead, the term **atypical pathogen** is preferred.
 - **Atypical pathogens** (eg Mycoplasma pneumoniae, C pneumoniae, Coxiella burnetii, C psittaci) are usually sensitive to antibiotics other than beta-lactams such as macrolides or fluoroquinolones which act intracellularly where these organisms replicate.
 - **Legionella species** share some characteristics but are not considered atypical as there are different species which can be acquired both in the community and hospital environment.

DEFINITION

- Hospital diagnosis is based on **new radiographic changes** on a chest radiograph in addition to symptoms and signs suggestive of pneumonia.
- Recognised features of pneumonia include fever, dyspnoea, pleuritic chest pain, productive cough, tachypnoea and focal crepitations or bronchial breathing on chest auscultation. *However, no prediction rules have shown reliable accuracy for diagnosing pneumonia in the absence of a chest radiograph.* The 2009 BTS guidelines propose the following definitions for community acquired pneumonia:
 - **Patients managed in the community (without a chest radiograph)**
 - Symptoms of an acute lower respiratory tract illness (cough and at least one other lower respiratory tract symptom eg. breathlessness, pleuritic chest pain)
 - and new focal chest signs on examination
 - and at least one systemic feature (either a symptom complex of sweating, fevers, shivers, aches and pains and/or temperature of 38°C or more)
 - and no other explanation for the illness, which is treated as CAP with antibiotics.
 - **Patients admitted to hospital (with a chest radiograph)**
 - Symptoms and signs consistent with an acute lower respiratory tract infection associated with new radiographic shadowing for which there is no other explanation (e.g. not pulmonary oedema or infarction); and the illness is the primary reason for hospital admission and is managed as pneumonia.

CLINICAL ASSESSMENT

History

- o Although a confident diagnosis of pneumonia cannot be made on the basis of history alone it is unlikely that the patient with **none** of the following symptoms will have pneumonia: Fever, Productive cough, Sweating, Shivering, Myalgia, Dyspnoea, Pleuritic chest pain...
- o A Focused **"AMPLE"** history should be able to identify factors which may influence the management plan if a diagnosis of pneumonia is made.
- o Only 20% of UK cases of Chlamydia psittaci pneumonia have a history of bird contact
- o Less than 10% of cases of Coxiella burnetii have a history of occupational exposure to animal sources (usually sheep).
- o Less than a quarter of legionella cases occur in clusters.

Examination

- o Identify features consistent with pneumonia eg bronchial breathing, dullness to percussion, crepitations on auscultation of the chest, pyrexia, tachypnoea and tachycardia.
- o Alternatively, the findings on chest examination may point to an alternative diagnosis such as pulmonary oedema, pleural effusion or pneumothorax.
- o As a minimum, the following parameters should be recorded: Temperature, Blood pressure, Pulse, Respiratory rate, Oxygen saturations

INVESTIGATION STRATEGIES

- o **ECG:** Pneumonia is not associated with specific ECG changes though sinus tachycardias are common. In the elderly, it is not uncommon for pneumonia to trigger atrial fibrillation or rate related ischaemia.
- o **Blood:** FBC, U&E, CRP, LFT
- o **Arterial Blood Gases/Lactate:** check if Sat <94% and for Lactate level

Microbiological tests

- o Do not routinely offer microbiological tests to patients with low-severity community-acquired pneumonia.
- o For patients with moderate- or high-severity community-acquired pneumonia:
 - Take blood and sputum cultures **and**
 - Consider pneumococcal and legionella urinary antigen tests.

- **CT chest:** Has no role in the routine investigation of patients with CAP.

Chest radiograph

- o The chest radiograph is the single most useful test available in the ED for diagnosing pneumonia. It is also in the identification of alternative diagnoses such as pulmonary oedema and exclusion of others e.g. pneumothorax.
- o Chest radiographs are not necessary on patients discharged from the ED with a diagnosis of CAP unless the diagnosis is in doubt or the patient is considered at risk of underlying pathology such as lung cancer.

- The following signs may help to confirm a diagnosis of pneumonia:

A. SILHOUETTE SIGN

- o The silhouette sign refers to the loss of the normal border between structures e.g.:
 - **Middle lobe pneumonia**, where the right heart margin is classically lost
 - **Right lower lobe pneumonia**, where the border of the diaphragm on the right side is obscured, while the right heart margin remains clear

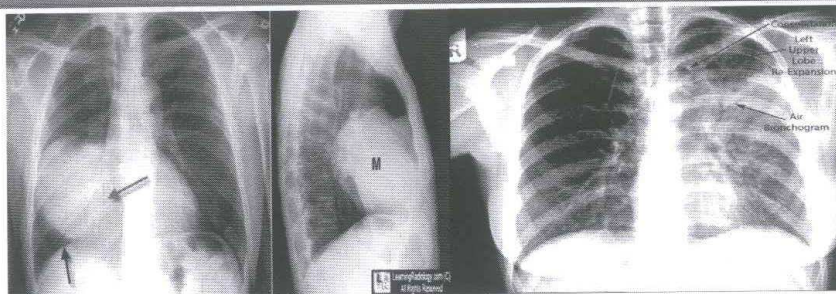


Figure 1.10. 1. Silhouette sign and Airbronchogram

B. AIR BRONCHOGRAM

- As the bronchi branch a point is reached where the cartilaginous bronchial walls are too thin to visualise (unless calcified) and it is not possible to distinguish air in the bronchi from air in the surrounding alveoli.
- However, if the surrounding alveoli fill with fluid or pus then branching radiolucent air passages (air bronchograms) may be seen.

CURB-65 SCORE

Symptom	Points
Confusion	1
Urea >7 mmol/l	1
Respiratory rate ≥ 30	1
BP: SBP < 90 mmHg, DBP ≤ 60 mmHg	1
Age ≥ 65	1

• 0 to 1 < 5% mortality	• 0-1: Treat as an outpatient
• 2 to 3 < 10% mortality	• 2: Consider a short stay in hospital or watch very closely as an outpatient
• 4 to 5: 15-30% mortality	• 3-5: Requires hospitalization with consideration as to whether they need to be in the intensive care unit

- Other factors suggesting a need for admission irrespective of their CURB-65 score:**

- Hypoxaemia ($\text{SaO}_2 < 94\%$ or $\text{PaO}_2 < 8 \text{ kPa}$) regardless of FiO_2 .
- Bilateral or multi-lobe involvement on the chest radiograph.
- Presence of a co-existing disease e.g. CCF, chronic renal failure
- Age over 50 years
- Social admissions in elderly with no adverse factors (other than age).

MANAGEMENT OF CAP IN THE ED

- General management**

- Patients should be given the following advice: **Rest, Drink plenty of fluids, Stop smoking.**
- Patients discharged from the ED should be advised to see their GP for review **within 48 hours** or sooner if clinically indicated.
- Oxygen:** if the oxygen saturations < 94% on air or $\text{PaO}_2 < 8 \text{ kPa}$.
- Steroids:** not recommended in the routine treatment of pneumonia of any severity.

SPECIFIC MANAGEMENT

1. ANTIBIOTIC THERAPY

- Offer antibiotic therapy as soon as possible after diagnosis, and certainly **within 4 hours**, to patients with **hospital-acquired pneumonia**.

• LOW-SEVERITY COMMUNITY-ACQUIRED PNEUMONIA

- Offer a **5-day course of a single antibiotic** to patients with low-severity community-acquired pneumonia.
 - **Amoxicillin 500mg Po Tds X5/7 (IV if PO not possible)**
 - **Penicillin allergic: Clarithromycin 500 mg PO bid or Doxycycline 200 PO mg stat then 100 mg PO**
- Consider extending the course of the antibiotic for longer than 5 days as a possible management strategy for patients with low-severity community-acquired pneumonia whose symptoms do not improve as expected after 3 days.
- *Do not routinely offer patients with low-severity community-acquired pneumonia:*
 - *A fluoroquinolone*
 - *Dual antibiotic therapy.*

• MODERATE- SEVERITY COMMUNITY-ACQUIRED PNEUMONIA

- Consider a **7- to 10-day course** of antibiotic therapy for patients with moderate- or high-severity community-acquired pneumonia.
- Consider **dual antibiotic therapy** with **Amoxicillin and a Macrolide** for patients with moderate-severity community-acquired pneumonia
 - **Amoxicillin 500mg-1g Po Tds + Clarithromycin 500 mg PO bid (IV if PO not possible)**

• HIGH-SEVERITY COMMUNITY-ACQUIRED PNEUMONIA

- **Co-amoxycylav 1.2g IVI tds + Clarithromycin 500mg bid IV**
- **Add Levofloxacin 500mg PO/IV OD: if Legionella suspected.**
- **Penicillin allergy:**
 - **Not IgE mediated reaction/Anaphylaxis: Cefuroxime 750mg-1.5g TDS IV + Clarithromycin 500mg bid IV**
 - **Severe IgE mediated reaction: Levofloxacin 500mg PO/IV OD (12 hly if severe)**

• Glucocorticosteroid treatment

- Do not routinely offer a glucocorticosteroid to patients with community-acquired pneumonia unless they have other conditions for which glucocorticosteroid treatment is indicated.

PATIENT INFORMATION

- Explain to patients with community-acquired pneumonia that after starting treatment their symptoms should steadily improve, although the rate of improvement will vary with the severity of the pneumonia, and most people can expect that by:
 - **1 week:** fever should have resolved
 - **4 weeks:** chest pain and sputum production should have substantially reduced
 - **6 weeks:** cough and breathlessness should have substantially reduced
 - **3 months:** most symptoms should have resolved but fatigue may still be present
 - **6 months:** most people will feel back to normal.
- Advise patients with community-acquired pneumonia to consult their healthcare professional if they feel that their condition is deteriorating or not improving as expected.

IV. SPONTANEOUS PNEUMOTHORAX

INTRODUCTION

- A pneumothorax is a collection of gas in the pleural space that results in a variable amount of lung collapse on the affected side.
- By definition, spontaneous pneumothoraces occur in the absence of any trauma (including iatrogenic causes) to the chest wall.

1. PRIMARY SPONTANEOUS PNEUMOTHORAX

- Occurs in people with no underlying lung pathology.
- The classic presentation is that of **sudden onset of pleuritic chest pain and dyspnoea at rest**.
- The symptoms do not correlate closely with the size of the pneumothorax.
- In many cases the symptoms are mild and approximately half of patients will present after more than 2 days of symptoms.

2. SECONDARY SPONTANEOUS PNEUMOTHORAX

- Occurs in patients with pre-existing lung parenchymal or pleural pathology (e.g. asthma, lung carcinoma).
- The symptoms are often more severe than those associated with a primary pneumothorax because lung function may already have been compromised by the underlying pathological process.
- The symptoms will vary depending on the cause e.g. fever, weight loss, night sweats but the primary complaint is that of breathlessness which is often out of proportion to the size of the pneumothorax radiologically.
- Unlike symptoms, the examination findings in primary spontaneous pneumothoraces are affected by the size of the pneumothorax. A small pneumothorax can be impossible to identify on clinical examination.
- If the pneumothorax is large, then some of the following features may be present:
 - Tachycardia and Tachypnoea
 - Reduced breath sounds on the affected side
 - Reduced chest expansion on the affected side as the patient splints the chest wall
 - Hyper-resonance on the affected side
 - Decreased tactile / vocal fremitus on the affected side
- The diagnosis is usually confirmed radiologically, following which specific information should be sought in order to guide management, advice and appropriate patient disposition/ follow-up.

3. TENSION PNEUMOTHORAX

- If the pleural leak exerts a one-way valve effect, then a tension pneumothorax can develop.
- This recognition and management of this complication is discussed later in the session. (Refer Major Trauma and fractures section, Chapter VI: Thoracic Trauma)

Information required for planning management and follow up for a patient with a spontaneous pneumothorax

Age of the patient

Does the patient feel breathless?

Determine if the pneumothorax is primary or secondary by reviewing the patients:

- Past medical history and Medication
- History of presenting complaint (specifically ask about trauma)
- Chest radiograph

History of previous pneumothorax (side, size and treatment)

Classify the size of the pneumothorax from the chest radiograph

- Small $\leq 2\text{cm}$
- Large $> 2\text{cm}$

Duration of symptoms

Smoker (and how many cigarettes they smoke per day)

Family history of pneumothorax

Vocation

Plans for holidays/ hobbies involving flying or SCUBA diving

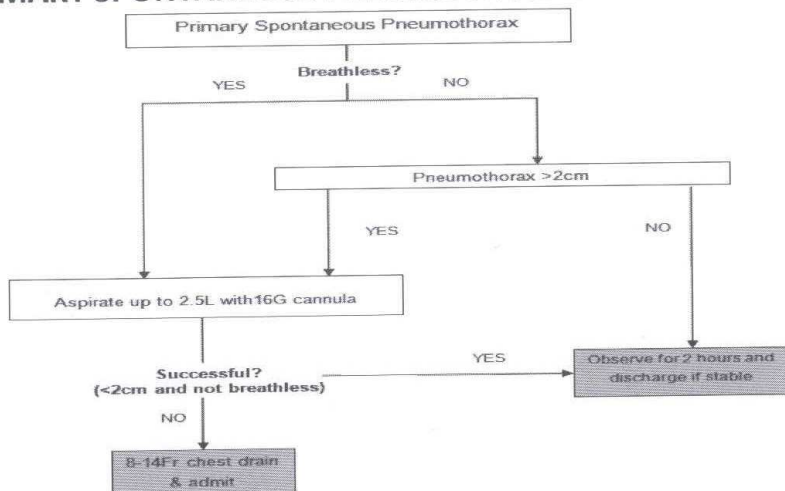
INVESTIGATION STRATEGIES

- **Radiographs**
 - The most useful investigation is the **PA chest radiograph**.
- **CT Scan**
 - CT is considered the **gold standard** at identification of a pneumothorax and is particularly valuable when radiographs are difficult to interpret or specific drain placement is required eg bullous lung disease, loculated pneumothoraces, and surgical emphysema.
- **Ultrasound**
 - Ultrasound show promise with reports of sensitivities at identifying pneumothoraces in trauma and post procedure (eg lung biopsy) patients of ~95%.
- **Arterial blood gases**
 - Arterial gas monitoring may demonstrate **hypoxia** but the information gained is unlikely to alter the management plan.
 - Their main use is when administering supplemental oxygen to patients with pneumothoraces secondary to COPD.

MANAGEMENT OF SPONTANEOUS PNEUMOTHORAX IN THE ED

- *Management depends upon whether the patient is **symptomatic**, whether the pneumothorax is **primary or secondary**, and its **size** on the PA radiograph.*
- **Supplemental oxygen:**
 - A pneumothorax will resolve up to **4 times faster** if high flow oxygen is administered.
 - Symptomatic patients and those admitted for observation should have high flow oxygen administered (**15l/min via a non re-breathe mask with a reservoir**).
 - **Entonox diffuses into air spaces and can convert an uncomplicated pneumothorax into a tension pneumothorax.**
 - *Its use as an analgesic is contraindicated in this setting.*

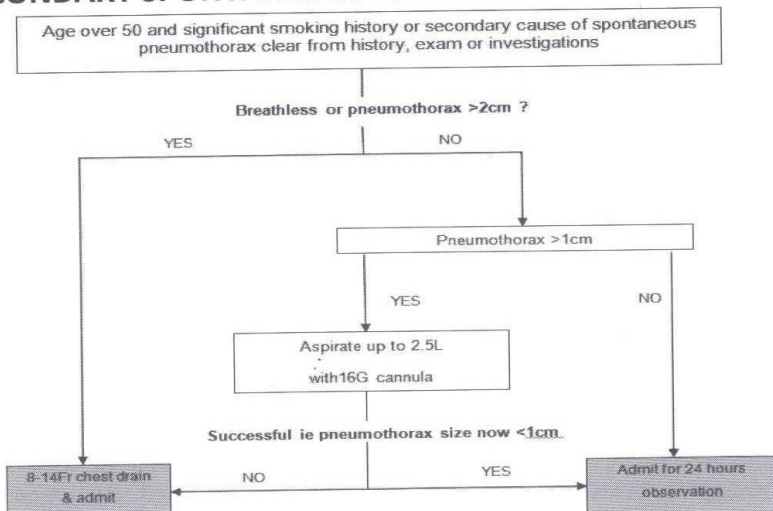
PRIMARY SPONTANEOUS PNEUMOTHORACES



All patients admitted should be given high flow oxygen

Discharged patients should be given verbal & written advice and have a follow up appointment made with a respiratory specialist in 2-4 weeks

SECONDARY SPONTANEOUS PNEUMOTHORACES



All patients require admission and should be prescribed high flow oxygen or controlled oxygen if at risk of oxygen sensitivity

KEY LEARNING POINTS

- **Smoking** is strongly associated with pneumothorax recurrence
- **Breathless patients** require intervention regardless of pneumothorax size
- **All patients with secondary pneumothoraces** require admission
- **Oxygen** should be applied to all patients with a pneumothorax if they are breathless or require admission
- Without supplemental oxygenation, spontaneous pneumothoraces resolve at a rate of approximately 2% of the hemi-thorax volume per day.
 - **A 1cm pneumothorax** (~25% pneumothorax) would be expected to fully resolve in approximately 12 days.
 - **A 2cm pneumothorax** (~30-50% pneumothorax) may take 3-4 weeks to fully resolve.
- Aspiration should be performed until the patient **coughs; no more can be aspirated** or when **2.5 mls have been aspirated**.
- Simple (needle) aspiration should be considered the first-line treatment for primary spontaneous pneumothoraces that require intervention
- Simple (needle) aspiration should be only be used for secondary pneumothoraces when the pneumothorax is small (1-2cm) and the patient is not breathless
- Small drains are as effective as large drains in treating spontaneous pneumothoraces and their use is preferred
- Patients discharged from the ED following a spontaneous pneumothorax should ideally be reviewed by a **respiratory physician after 2 weeks**. In practice, it may be impossible to access specialist clinics in the recommended timeframe. If this is the case, then the patient should be advised to initially return to the ED, at 2 weeks, **for a repeat chest radiograph and senior doctor review pending specialist review**.
- **Important advices**
 - **Smokers** should be advised to quit and seek assistance from their GP to successfully achieve this.
 - **Patients should not fly** until a week has elapsed since complete resolution of the pneumothorax has been demonstrated on a chest radiograph or until they have recovered from a definitive surgical procedure aimed to prevent pneumothorax recurrence.
 - **Patients should never dive** after a pneumothorax unless bilateral surgical pleurectomy has been performed.
- If the pneumothorax is recurrent, or the patient has a high-risk vocation, referral for a cardiothoracic outpatient appointment is appropriate.
- **Refer to cardiothoracic surgeon if:**
 1. Second ipsilateral pneumothorax
 2. First contra-lateral pneumothorax
 3. Bilateral spontaneous pneumothorax
 4. Persistent air leak or failure of lung re-expansion 5 days after chest drain insertion
 5. Spontaneous haemothorax
 6. Professions at risk (e.g. pilots, divers)
 7. Pregnancy

V. PLEURAL EFFUSION

INTRODUCTION

- A pleural effusion is an abnormal collection of fluid within the pleural space.
- More commonly, pleural effusions are found incidentally on chest radiographs requested for another acute problem (e.g. heart failure, pneumonia) or a chronic condition already known to the patient (e.g. malignant effusion).

AETIOLOGY

- **Congestive cardiac failure** is the commonest cause of pleural effusion.
- Normal pleural fluid is formed from the filtration of plasma by healthy parietal pleural membranes and has the following characteristics:
 - A glucose content similar to plasma
 - A low sodium content (1-2 g/dl)
 - A low white cell count of <1000 cells / mm³
 - A low lactate dehydrogenase (LDH) level ($<50\%$ that of plasma)

1. TRANSUDATES

- Transudates are associated with **increased systemic or pulmonary capillary hydrostatic pressure** (e.g. congestive cardiac failure) or **decreased colloid osmotic pressure** (e.g. hypoalbuminaemia, cirrhosis). These factors often co-exist.
- In conditions resulting in the formation of transudates, **the pleural membranes are intact and the permeability of pleural capillaries to proteins is normal**.
- Neither is involved in the pathogenesis of fluid formation.

2. EXUDATES

- The formation of exudates is associated with **altered permeability of pleural membranes, increased capillary wall permeability to proteins or vascular disruption**.
- It can also be associated with **reduced or obstructed lymphatic drainage** from the pleural space.
- **Disease of the pleural membranes** is involved in the formation of exudates and increased capillary permeability to protein results in elevated protein content of the fluid.

CLINICAL ASSESSMENT

- **History**
- **Classical symptoms and signs**
 - Dyspnoea, stony dullness to chest percussion, reduced breath sounds, reduced tactile fremitus, and asymmetric chest expansion.
- **Non-specific features**
 - Chest pain, upper abdominal pain, shoulder tip pain, peripheral oedema, haemoptysis, evidence of malignancy.
 - **Patients with chest pain and pleural effusion are more likely to have an exudative aetiology such as pleural infection, pulmonary infarction (PE) or malignancy.**
- **EMERGENCY MANAGEMENT OF THE UNSTABLE PATIENT**
 - Patients with pleural effusions do not classically present with sudden breathlessness or cardiac instability.
 - Unless signs consistent with a massive pleural effusion are present then other co-existent pathologies should be considered.

- o Pulmonary embolism or infarction cause dyspnoea out of all proportion to the size of any pleural effusion and Emergency physicians should have a high index of suspicion for this diagnosis in this setting.

1. MASSIVE PLEURAL EFFUSION WITH MIDLINE SHIFT

- As much as 3 litres of fluid can be contained within each side of the thorax. In this setting, the following treatment should be instituted:
 - o **A: Sit the patient upright** to improve their ventilation
 - o **B: Administer high flow oxygen**
 - o **C: Support the circulation** as indicated (e.g. intravenous crystalloid bolus)
 - In patients with likely transudates or bilateral effusions **treat the underlying cause aggressively** (such patients rarely need therapeutic aspiration of the pleural fluid).
 - In patients with unilateral massive pleural effusion +/- midline shift, **urgent therapeutic drainage** of the fluid is required.
 - **No more than 1.5 litres of fluid should be drained in the first hour** as re-expansion pulmonary oedema (which as a significant mortality risk) can result when greater volumes are drained.
 - A **small-bore chest drain** is usually all that is necessary.
 - Send fluid for **laboratory analysis**.

• INVESTIGATION STRATEGIES

- o **Chest Radiograph:** to identifying the size and location of the effusion and any underlying aetiology.
- o **Blood:** ABG, FBC, U&E, Serum Protein, Serum LDH, Serum Glucose, Serum Amylase
- o **Pleural fluid analysis:** The gross appearance of the fluid should be noted as this may suggest a specific diagnosis
 - Protein content
 - LDH level
 - Cytology, cell count and differential
 - Fluid pH/ Fluid glucose
 - Gram staining and culture

Pleural Fluid laboratory analysis

- o A **transudate** contains **less than 25 g/l of protein**
- o An **exudate** contains **more than 35 g/l of protein**
- If the pleural fluid contains protein at levels between **25 g/l and 35 g/l**, then **Lights Criteria** should be used to decide whether the effusion is a transudate or an exudate.

LIGHTS CRITERIA

- States that **the fluid is an exudate** if one or more of the following criteria are met:
 - o **Pleural fluid Protein: Serum protein** ratio is greater than **0.5 (Pfp/SP>0.5)**
 - o **Pleural fluid LDH: Serum LDH** is greater than **0.6 (PfldH/SLDH>0.6)**
 - o **Pleural fluid LDH** is greater than two thirds the upper limit of **normal serum LDH**. ($PfldH > \frac{2}{3}$ upper limit SLDH)

Advanced Imaging Studies

- **Ultrasound**
 - o The BTS strongly recommends the use of ultrasound to guide pleural aspiration
 - o *If ultrasound is not employed and the aspiration fails, no subsequent attempts should be made until imaging has been performed.*
- **CT Scanning:** useful in differentiating benign from malignant pleural effusion.

MANAGEMENT OF PLEURAL EFFUSION IN ED**1. THE STABLE PATIENT WITH A PLEURAL TRANSUDATE**

- o **Treat underlying cause:** CCF, cirrhosis of the liver, hypoalbuminaemia and hypothyroidism should be treated aggressively.
- o Diagnostic aspiration of pleural fluid is not required in most cases.
- o **Therapeutic drainage** if patient remains symptomatic despite treatment of the underlying cause.

2. THE STABLE PATIENT WITH PLEURAL INFECTION

- o More than half the patients admitted to hospital with pneumonia will have associated pleural fluid. The nature of the pleural fluid associated with pneumonia is key to guiding subsequent treatment.
- o In particular, a **low pH (<7.2)** found in pleural fluid is an indication for chest drainage.
- o All patients with signs of pneumonia and a pleural effusion should have diagnostic aspiration of pleural fluid with fluid analysis, unless their condition makes this an inappropriate intervention.
- o This may be performed in the ED or arranged via in-patient specialties.
- o In patients with poor respiratory reserve or with small or loculated collections ultrasound guidance should be used.
- o Patients with **simple para-pneumonic effusions** (see below) rarely require therapeutic drainage.
 - Normal fluid pH (pH >7.2) and glucose
 - Low WCC and no organisms
 - Can be **treated with antibiotics alone**
- o Patients with **complex para-pneumonic effusions** (see below) require therapeutic drainage.
 - Low fluid pH (pH <7.2) and glucose
 - Elevated WCC +/- organisms
 - **Small bore chest drains** are recommended in the first instance.
 - If patient fails to respond **intra-pleural fibrinolytic drugs** (e.g. urokinase) may be required.
 - Patients who do not respond to drainage and intra-pleural fibrinolytic drugs should be referred for **surgical intervention**.

3. THE STABLE PATIENT WITH MALIGNANT EFFUSION

- o **Stable patients:** Discussed with the medical team and may be discharged from the ED for urgent out-patient follow up.
- o **Symptomatic patients** will require therapeutic pleural drainage. **No more than 1.5 litres** should be drained at any one time.
- o Except for patients with very short life expectancy, pleural drainage should be combined with **pleurodesis** (e.g. with intrapleural tetracyclines, talc or bleomycin) as there is a high recurrence rate without this.
- o Some patients with fibrous or loculated effusions may also require **intrapleural fibrinolytic therapy** (e.g. with urokinase).
- o Other approaches to subsequent management include **thoracoscopy, intermittent therapeutic drainage** (usually those with short life expectancy), **long term indwelling pleural catheter** and **pleuro-peritoneal shunting**.

VI. CARDIOGENIC PULMONARY OEDEMA (CPO)

INTRODUCTION

- CPO is characterised by the presence of excess fluid within the pulmonary interstitium and, at its most severe, within the alveoli. It is due to a primary cardiac or circulatory cause rather than other forms of pulmonary oedema (eg. neurogenic pulmonary oedema).

CLINICAL ASSESSMENT

- Patients with CPO classically present to the ED in extremis, **often in the early morning**.
- The history is often limited initially and CPO can prove to be a difficult diagnosis to make, as it frequently occurs in patients with coexisting COPD and those at risk of pneumonia or pulmonary embolism.
- **History**
 - The history should focus on establishing the diagnosis of CPO as well identifying any precipitating event.
 - Previous episodes of CPO, orthopnoea or PND together with any historical factors that could point towards a precipitating cause should be elicited.

CLINICAL EXAMINATION

- **Airway**
 - Usually intact, unless conscious level impaired by hypoxia or hypercapnoea.
 - In extreme cases blood stained frothy sputum may be present
- **Breathing**
 - **Tachypnoea**: using accessory muscles, adapting posture to maximise air entry.
 - The **bases may be dull to percussion** as small **pleural effusions** are common
 - SpO₂ <90% on air
 - Auscultation reveals **bibasal inspiratory crepitations**.
 - In some cases, **wheeze** predominates which can confuse the picture.
- **Circulation**
 - Patients appear '**diaphoretic**' pale and their skin cold and clammy
 - **Sweats profusely**: Monitoring stickers and line adhesive may not stick
 - **Sinus tachycardia** is common but arrhythmias such as new atrial fibrillation may precipitate CPO.
 - **The blood pressure is usually high**; however, hypotension may be present and is associated with cardiogenic shock and increased mortality.
 - **The heart sounds may be inaudible** over the rales from the lungs but a gallop rhythm may be present. Murmurs, especially mitral regurgitation and aortic stenosis, should be listened for, and may reveal a precipitating cause.
 - **Level of hydration should be assessed**. Some patients may be **fluid overloaded** but many are **euvolaemic**. Assess the JVP, mucus membranes and urine output.
 - Look for peripheral oedema and hepatomegaly suggesting **right heart failure**.
- **Disability**
 - Initially patients are **alert and anxious**.
 - As their hypoxia worsens they may become **agitated**
 - With worsening respiratory failure, may become hypercapnoeic causing their conscious level to fall.
- **Exposure**
 - **Afebrile** with cold and clammy skin

TYPICAL CLINICAL FEATURES AND DIFFERENTIAL DIAGNOSIS OF CPO

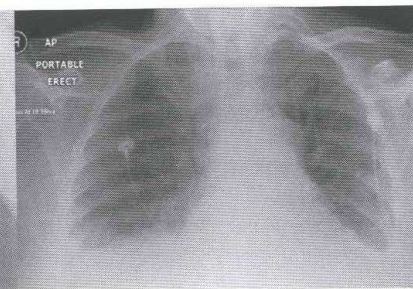
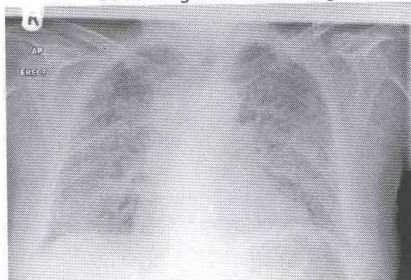
SIGN	CPO	COPD	Pneumonia	PE
Chest signs	Bilateral crepitations	Diffuse wheeze	Focal crepitations	Clear
Skin	Cold, pale, clammy	Dry and warm	Flushed, warm	Dry
Heart Sounds	Murmurs, 3 rd /4 th HS	Normal	Normal	Normal

INVESTIGATION STRATEGIES• **ECG**

- o It will often show a **tachycardia** and **possible left ventricular hypertrophy**.
- o It may reveal precipitating causes such as **ST segment changes** associated with an ACS (STEMI or NSTEMI) or an **arrhythmia** eg atrial fibrillation.

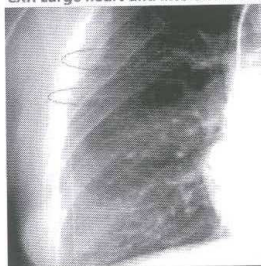
• **CXR**

- o Helpful in excluding other causes of breathlessness, such as pneumonia or pneumothorax. A normal CXR in the acutely short of breath patient would be more likely to suggest a pulmonary embolus or COPD/asthma.
- o **The chest X-ray in CPO can show:**
 - *Cardiomegaly and Upper lobe blood diversion,*
 - *KERLEY B septal lines,*
 - *Fluid in the interlobar fissures and Pleural effusions,*
 - *Bat's wing hilar shadowing*

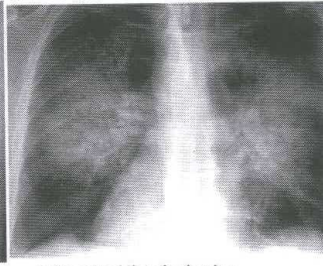
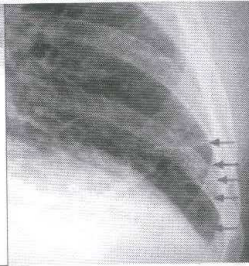


CXR Large heart and interstitial and alveolar oedema

CXR Bilateral pleural effusions & upper lobe diversion



Kerley B lines



Bat's wing hilar shadowing

• **Arterial Blood Gas**

- o **Hypoxaemia: Type 1 respiratory failure;** this contrasts with COPD patients in extremis (who have type 2 respiratory failure).

• **Other Blood Tests:**

- o Baseline bloods including **FBC, U&Es, LFT, Troponin, BNP and INR**

ED MANAGEMENT OF CARDIOGENIC PULMONARY OEDEMA

- Treatment should consist of **sitting the patient up**, administering **high flow O₂**, **intravenous nitrates** and instituting **NIV** if appropriate.

A Sit the patient Up

B **High flow O₂**: 15L/minute with a reservoir bag
NIV:

- CPAP** Commence PEEP at 5-7.5 cm H₂O & increase up to 10cm as tolerated
- BiPAP**

C

- Nitrates 10-20mcg/min**; Increase the nitrate infusion every 3-5 min by 5-10 mcg/min as BP allows until improvement.
- Nitroprusside**: Cautious infusion at **0.3 mcg/Kg/min**
- Dobutamine** Infusion commenced at **2-3mcg/kg/min**
- Furosemide 20-40mg IV**

Drugs **Morphine** (Only small boluses) **2.5-5mg IV**

PRACTICAL ASPECTS RELATED TO NON-INVASIVE VENTILATION IN CPO

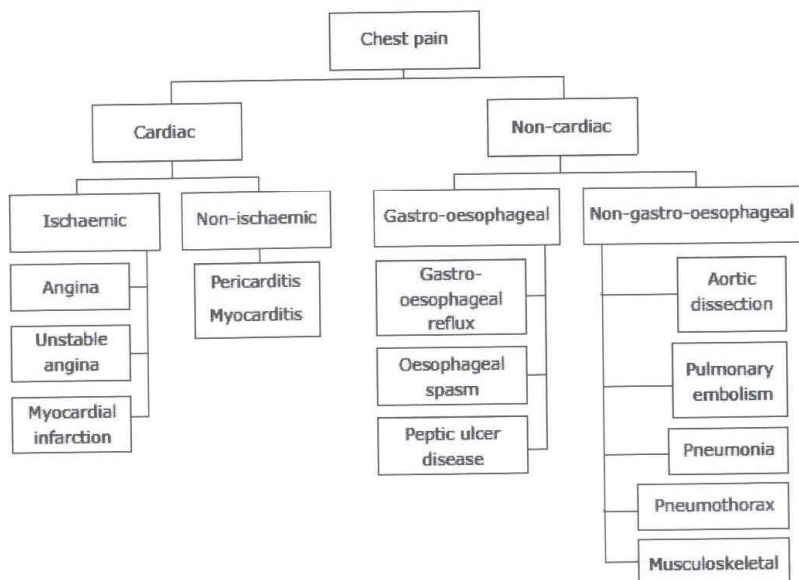
Indications	Consider in all patients with CPO Particularly pH<7.35 Respiratory rate >20/min
Cautions	Cardiogenic shock Agitated patient
"CARS"	Right ventricular failure Severe obstructive airways disease
Contraindications	Immediate endotracheal intubation indicated Respiratory arrest or inadequate spontaneous ventilation Worsening life threatening hypoxia Unconscious patient unable to protect own airway
How to deliver NIV	Correctly fitting mask Supplemental O ₂ Commence PEEP at 5-7.5 cm H ₂ O and increase to 10cm as tolerated Continue for 30min/hr until reduction in dyspnoea and saturations are maintained off NIV
Complications	Intolerance due to anxiety, skin/eye discomfort, dry mouth Worsening right ventricular failure Hypercapnoea Pneumothorax Aspiration

PROGNOSIS

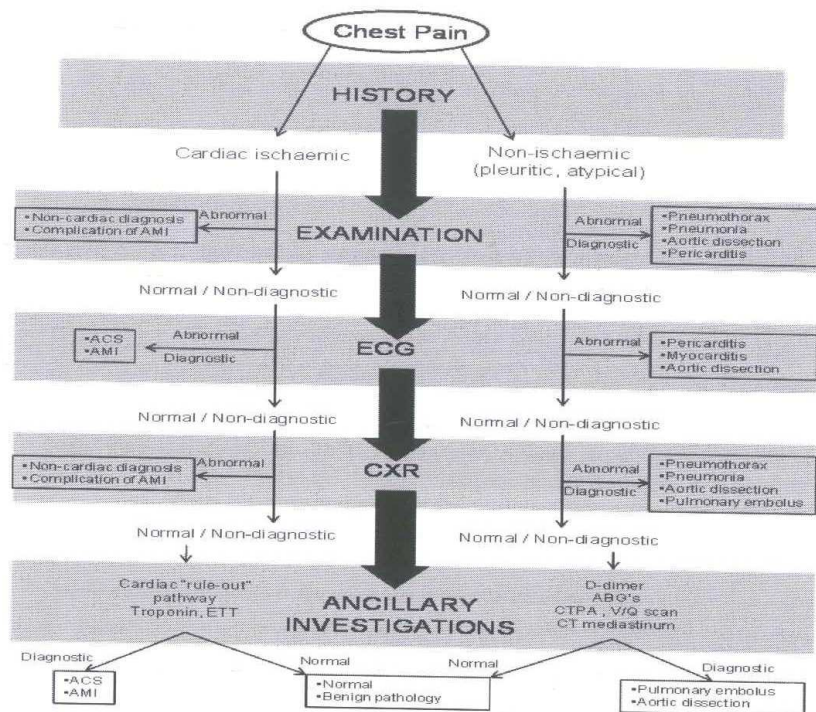
- The following features are associated with a worse outcome:
 - Advanced Age
 - Hypotension
 - Precipitated by ischaemia
 - Low Left Ventricular Ejection Fraction
 - Previous hospitalisation for heart failure
 - Wide QRS
 - Hyponatraemia
 - Marked BNP elevation/ Elevated troponin

CHAPTER 11. CHEST PAIN**I. CHEST PAIN SYNDROMES****1. THE SPECTRUM OF PATHOLOGY PRESENTING WITH CHEST PAIN**

SYSTEM	LIFE-THREATENING	URGENT	NON-URGENT
Cardiovascular	AMI Aortic dissection PE	Unstable angina Coronary vasospasm Pericarditis Myocarditis	Stable angina Valvular heart disease Hypertrophic cardiomyopathy
Pulmonary	Tension pneumothorax	Simple pneumothorax	Viral pleurisy Pneumonia
Musculoskeletal			Costochondritis Chest wall injury
Gastrointestinal	Oesophageal rupture	Pancreatitis	Cholecystitis Oesophageal reflux Biliary colic Peptic ulcer
Other		Mediastinitis	Postherpetic neuralgia Herpes zoster Malignancy Psychological/anxiety

2. CARDIAC AND NON-CARDIAC CAUSES OF CHEST PAIN**Cardiac and non-cardiac causes of chest pain**

3. DIAGNOSTIC APPROACH TO CHEST PAIN IN ED



4. Characteristic description of symptoms associated with major causes of chest pain

CONDITION	DESCRIPTION OF SYMPTOMS
Ischaemic cardiac pain	<ul style="list-style-type: none"> • Retrosternal 'pressure', 'tightness', 'constricting' • Radiation to shoulders/arms/neck/jaw • Crescendo in nature, related to exertion • Associated with diaphoresis, sweating, nausea, pallor
Pericarditis	<ul style="list-style-type: none"> • Atypical, retrosternal, sometimes pleuritic • Positional relieved on sitting forward
Gastro-oesophageal	<ul style="list-style-type: none"> • Retrosternal, 'burning' • Associated with ingestion
Aortic dissection	<ul style="list-style-type: none"> • 'Tearing' pain, sudden in onset, • Radiation to back
Pulmonary embolism	<ul style="list-style-type: none"> • Atypical, may be pleuritic • Associated with breathlessness; occasional haemoptysis
Pneumothorax	<ul style="list-style-type: none"> • Atypical, may be pleuritic • Associated with cough, sputum, fever
Musculoskeletal	<ul style="list-style-type: none"> • Sharp, positional, pleuritic • Aggravated by movement, deep inspiration and coughing

5. RISK FACTORS ASSOCIATED WITH MAJOR LIFE-THREATENING CAUSES OF CHEST PAIN

CONDITION	RISK FACTORS
Acute coronary syndromes	<ul style="list-style-type: none"> • Previous known coronary artery disease (previous myocardial infarction, angioplasty, etc.) • Positive family history • Advanced age, male gender • Diabetes, Hypertension, Hypercholesterolaemia • Active smoker, Obesity, Sedentary Lifestyle • Aspirin usage
Aortic dissection	<ul style="list-style-type: none"> • Chronic hypertension • Inherited connective tissue disorder, e.g. Marfan syndrome, Ehlers-Danlos syndrome • Bicuspid aortic valve/ Coarctation of the aorta • Pregnancy • Inflammatory aortic disease, e.g. Giant Cell Arteritis
Pulmonary embolism	<ul style="list-style-type: none"> • Previous history of venous thromboembolic disease • Pregnancy or puerperium • Positive family history of venous thromboembolic disease (two or more family members) • Recent prolonged immobilisation (>3 days) • Major surgery within previous 12 weeks • Fracture of lower limb within previous 12 weeks • Active cancer (within previous 6 months, recent treatment, palliation) • Lower extremity paralysis

DIAGNOSIS	PHYSICAL FINDINGS
ACS	Diaphoresis, tachycardia, tachypnoea, pallor
Complications of acute MI	Hypotension, third heart sound, pulmonary crepitations, elevated JVP, bradycardia, new murmur
Aortic dissection	Diaphoresis, hypotension, hypertension, tachycardia, differential blood pressures and/or pulses, new murmur (aortic regurgitation), focal neurological findings
Pulmonary embolism	Acute respiratory distress, diaphoresis, hypotension, tachycardia, hypoxaemia, elevated JVP, pleural rub
Pneumonia	Fever, signs of pulmonary collapse/consolidation , tachycardia, tachypnoea
Oesophageal rupture	Diaphoresis, hypotension, tachycardia, Fever, Hamman's sign*, subcutaneous emphysema , epigastric tenderness
Simple pneumothorax	Tachypnoea, tachycardia, unilateral diminished air entry and breath sounds , subcutaneous emphysema
Tension pneumothorax	Tachypnoea, hypotension, tachycardia, hypoxaemia, elevated JVP, unilateral diminished air entry and breath sounds , subcutaneous emphysema, tracheal deviation
Pericarditis	Tachycardia, Fever, Pericardial rub
Myocarditis	Hypotension, tachycardia, fever, third heart sound , pulmonary crepitations, displaced apex beat
Mediastinitis	Tachycardia, fever, Hamman's sign* , subcutaneous emphysema, hypotension
Cholecystitis	Diaphoresis, fever, tachycardia, right upper quadrant tenderness

6. ECG FINDINGS ASSOCIATED WITH NON-ISCHAEMIC CHEST PAIN CONDITIONS

ECG finding	Context	Diagnosis
Diffuse concave-upward ST segment elevation	Positional pain Pericardial rub	Pericarditis
Right ventricular strain pattern	Pleuritic pain Hypoxia Pleural rub	P.E.
Diffuse ST/T wave changes	Atypical pain Heart failure	Myocarditis
Inferior ST elevation	Tearing chest pain Radiation to back Differential pulses Differential blood pressures New diastolic murmur	Aortic dissection

7. RADIOGRAPHIC FINDINGS IN CONDITIONS PRESENTING WITH CHEST PAIN

Condition	Radiographic finding	Comment
ACS	No specific radiographic finding	
Aortic dissection	Mediastinal widening Abnormal aortic contour Globular heart shadow Pleural effusion (haemothorax)	Suggestive in context Unusual finding Rare finding Rare finding
Pneumothorax	Absence of pulmonary vascular markings	Diagnostic
Tension pneumothorax	Absence of pulmonary vascular markings Mediastinal displacement	Diagnostic Diagnostic
Pneumonia	Localised or diffuse pulmonary infiltration Localised pulmonary atelectasis Localised Consolidation	Diagnostic in context Diagnostic in context
Pulmonary embolism	Normal chest radiograph Localised pulmonary atelectasis Small pleural effusion	Suggestive in context Rare finding Rare finding
Oesophageal rupture	Pneumomediastinum	Diagnostic in context
Mediastinitis	Pneumomediastinum	Diagnostic in context
Pericarditis	Globular heart shadow	Pericardial effusion
Myocarditis	Enlarged cardiac shadow	Dilated cardiomyopathy

8. ANCILLARY INVESTIGATIONS

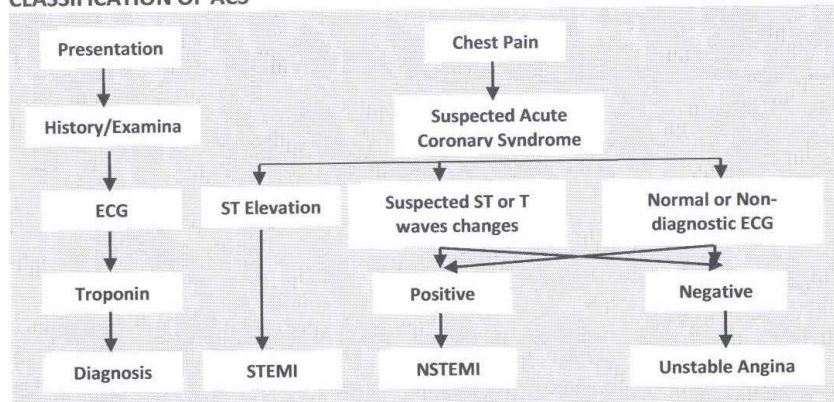
- The history, physical examination, ECG and CXR will normally allow the emergency physician to be fairly confident to achieve a diagnosis in a patient with chest pain presenting to the ED.
- ACS:
 - Cardiac markers (e.g. troponin) and possible exercise testing.
- Pulmonary embolism
 - For patients at low risk: **D-dimer assay**.
 - For patients at intermediate or high risk: **Ventilation perfusion (V/Q) scan or CT Pulmonary Angiogram (CTPA)**.
- Aortic dissection:
 - CT Mediastinum** (to definitively exclude aortic dissection)

II. ACUTE CORONARY SYNDROMES

INTRODUCTION

- Acute coronary syndromes (ACS) encompass a broad range of presentations including
 - Unstable angina (UA),
 - Non-ST-segment elevation myocardial infarction (NSTEMI)
 - ST-segment elevation myocardial infarction (STEMI).
- Myocardial infarction** is defined pathologically as myocardial cell death following prolonged ischaemia.
- The criteria for acute, evolving or recent myocardial infarction are as shown below:

CLASSIFICATION OF ACS



PATHOPHYSIOLOGY OF ACS

- Acute Coronary Syndromes occur when myocardial oxygen demand exceeds circulatory supply. This initially results in **ischaemia**; prolonged ischaemia results in **infarction** (myocardial cell necrosis).
- A reduction in oxygen supply is precipitated by mechanical or inflammatory disruption (rupture or erosion) of an atherosclerotic coronary artery plaque associated with varying degrees of local vasoconstriction, thrombosis and microembolisation.
- Atherosclerotic plaque disruption** initiates **thrombosis** with platelet activation and platelet aggregation. Thrombus formation **in the context of STEMI is fibrin-rich**; it causes coronary artery occlusion leading to myocardial ischaemia and subsequent infarction. This will manifest electrocardiographically as **ST segment elevation** with a distribution of changes depending upon the coronary artery affected.
- Thrombus occurring **in the context of NSTEMI-ACS is platelet-rich**; spontaneous thrombolysis and fragmentation into smaller particles release platelet emboli, which may cause small areas of more distal infarction (micro-infarction) without complete occlusion of the coronary artery.
- It is the process thought to be occurring in infarction without ST elevation (i.e. **NSTEMI**).
- The thrombotic response to plaque disruption is a dynamic process of thrombosis and thrombolysis, mediator induced vasoconstriction, and varying degrees of platelet aggregation and embolisation.
- Which particular process predominates determines the clinical syndrome (i.e. STEMI, NSTEMI or UA), and, in turn, the most appropriate subsequent therapy.

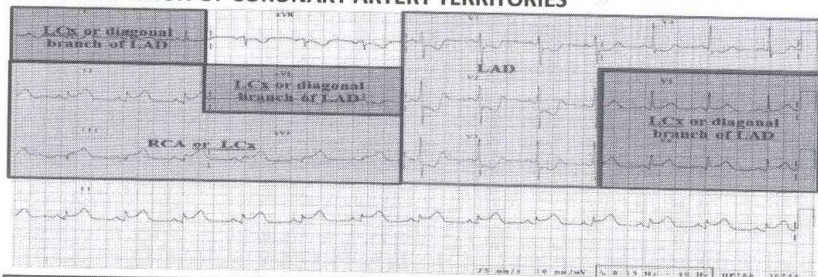
CLINICAL ASSESSMENT

- **History**
- The classic presenting symptom of ACS is **chest pain**, which is traditionally described as having a characteristic nature:
 - Heavy, aching or tight
 - Central chest or left sided
 - Not related to respiration or movement
 - May radiate to one or both arms, neck, or jaw
- Atypical presentations of ACS are common, occurring in up to **33% of patients**, mostly in the elderly, diabetics and women.
- Advanced age, co-morbid factors, delay in diagnosis, delayed or reduced use of reperfusion therapy, and reduced use of adjuvant therapies all contribute to the increased mortality in this population.

INVESTIGATION STRATEGIES

- **Electrocardiography**
- Broadly speaking, the ECG will directly determine whether a patients further management follows:
 - An immediate fibrinolytic or mechanical reperfusion strategy (i.e. ST elevation)
 - An anti-thrombotic and anti-platelet strategy (ST depression or T wave inversion)
 - A rule out strategy (normal ECG)
- The following ECG changes are indicative of myocardial ischaemia that may progress to AMI:
 - Patients with **ST segment elevation in two or more contiguous leads** (greater than 0.2mV in leads V1, V2, or V3 and greater than 0.1mV in other leads)
 - Patients with **ST segment depression**
 - Patients with **T wave abnormalities only**

ECG LOCALISATION OF CORONARY ARTERY TERRITORIES



Description	ECG Leads With Changes	Artery Occluded
Inferior	II, III and aVF	RCA
Anteroapical	V3 and V4	Distal LAD
Anteroseptal	V1 and V2	LAD
Anterolateral	I, aVL, V5 and V6	Circumflex Artery
Extensive Anterior	I, aVL and V2-V6	Proximal LCA
True Posterior	Tall R in V1	RCA

Left Anterior Descending (LAD), Left Circumflex (LCx) and Right Coronary Artery (RCA)

1. STEMI: ST-SEGMENT ELEVATION MI

- The presence of **ST segment elevation**, **new Q wave formation**, or a **new conduction deficit** (eg. **left bundle branch block**) in the context of acute ischaemic chest pain is associated with such significantly positive likelihood ratios for AMI that the diagnosis can usually be made with confidence and appropriate therapy commenced.
- However, **the ECG by itself cannot define AMI**, which also requires the demonstration of a cardiac marker rise.
- There are situations where this injury pattern (i.e. **ST segment elevation**) does not necessarily indicate that myocardial necrosis has or will occur:
 - *Aborted myocardial infarction where early reperfusion has occurred*
 - *Coronary artery vasospasm with spontaneous resolution.*
- ST segment elevation will typically be found in a territorial distribution on the ECG that reflects, and is determined by, coronary artery anatomy.

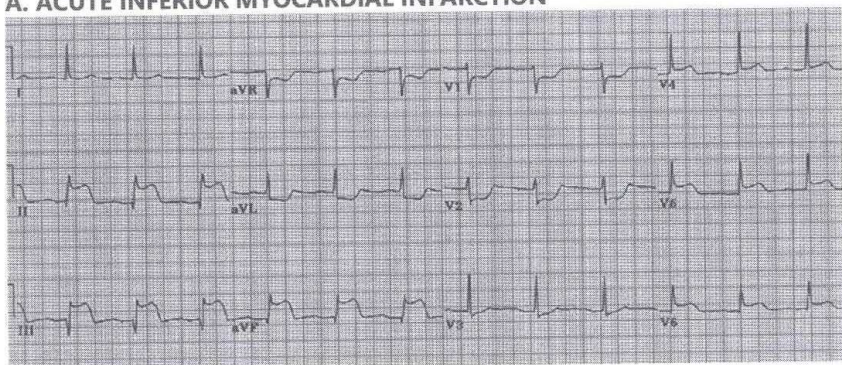
A. ACUTE INFERIOR MYOCARDIAL INFARCTION

Figure 1.11.1. ST segment elevation in the inferior leads (II, III, and aVF) with reciprocal ST segment depression in the anterior leads (V1, V2, and V3), possibly representing posterior extension of the infarct.

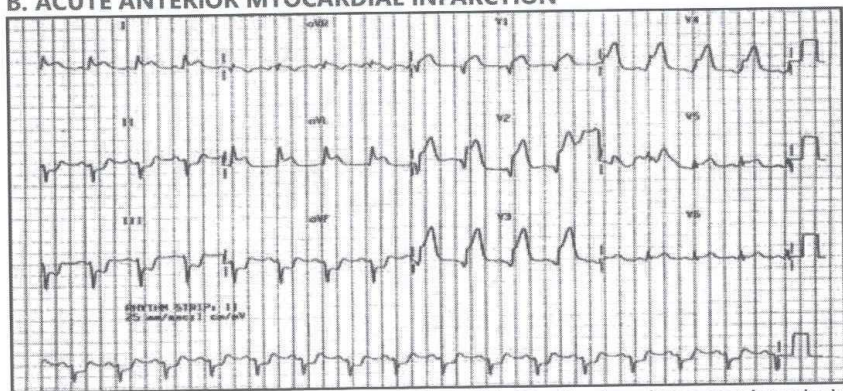
B. ACUTE ANTERIOR MYOCARDIAL INFARCTION

Figure 1.11.2. ST segment elevation across the anterior leads (V1 to V4) with reciprocal ST segment depression in the inferior leads (II, III, and aVF).

2. NSTEMI/UA: ST-SEGMENT DEPRESSION / T WAVE CHANGES

- The presence of **ST segment depression and/or T wave changes** in the context of acute ischaemic chest pain normally indicates myocardial ischaemia (i.e. unstable angina) but is also associated with a positive likelihood ratio for AMI (i.e. NSTEMI).
- Approximately 50% of patients with ST depression and 33% of patients with T wave inversion will subsequently be shown to have myocardial infarction.
- This group of patients are presenting with an ACS (i.e. UA or NSTEMI).

A. ACS WITH ST DEPRESSION AND T WAVE INVERSION

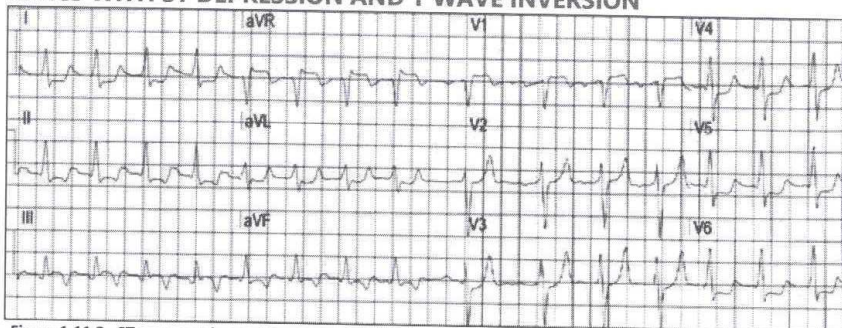


Figure 1.11.3. ST segment depression most evident in lateral leads (V4-V6, I, aVL) and T wave inversion in inferior leads (II, III and aVF).

3. ADDITIONAL CHEST LEADS

- Additional chest leads are required if posterior (**V7, V8, V9**) or right sided (**V4R**) infarction is suspected following a standard (12 lead) ECG specifically in:
 - Patients with **inferior ST segment elevation** because the majority of right sided and posterior infarcts occur as extensions of inferior infarcts. *This may affect management patients with right sided myocardial infarction and hypotension may respond to fluid resuscitation.*
 - Patients with **anteroseptal ST segment depression** (indicating ischaemia) because this may be masking true posterior infarction; this will, if demonstrated, affect immediate treatment.

A. ACUTE INFERIOR MYOCARDIAL INFARCTION (Standard 12 Lead ECG)

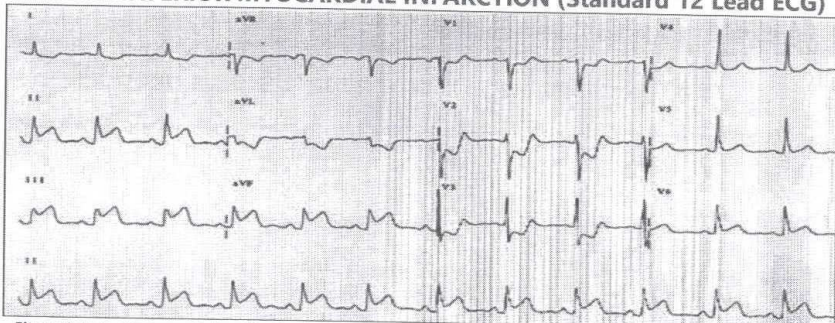


Figure 1.11.4. ST segment elevation in the inferior leads with reciprocal changes in the anteroseptal leads (V1-3)

B. POSTERIOR ST ELEVATION

Confirming Posterior Extension of Infarct (Additional Leads)

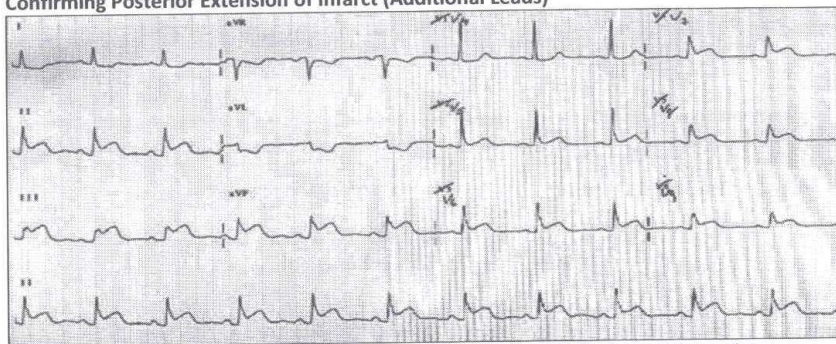


Figure 1.11.5. ST segment elevation in the posterior leads (V7-9) in the same patient as above

4. CHEST PAIN AND LBBB

- Patients with **ischaemic cardiac chest pain** and **left bundle branch block (LBBB)** should be assumed to be **having an AMI** and **should be considered for immediate reperfusion therapy**, since they have been shown to have amongst the highest mortality of patients with AMI, and also gain the greatest benefit from thrombolysis.
- ECG criteria have been identified that have good specificity (but poor sensitivity) for AMI in patients with LBBB:
- **ECG criteria suggesting AMI in LBBB (Sgarbossa criteria)**
 - ST elevation $>1\text{mm}$ in leads where the QRS complex is predominantly positive (i.e. V5, V6)
 - ST depression $>1\text{mm}$ in leads where the QRS complex is predominantly negative (i.e. V1, V2, V3)
 - ST elevation $>5\text{mm}$ in leads where the QRS complex is predominantly negative (i.e. V1, V2, V3)

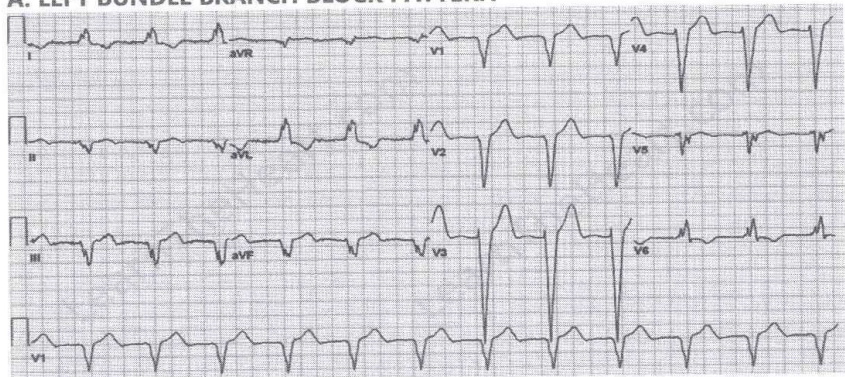
A. LEFT BUNDLE BRANCH BLOCK PATTERN

Figure 1.11.6. T waves are discordant with the QRS complexes; this is a standard LBBB morphology

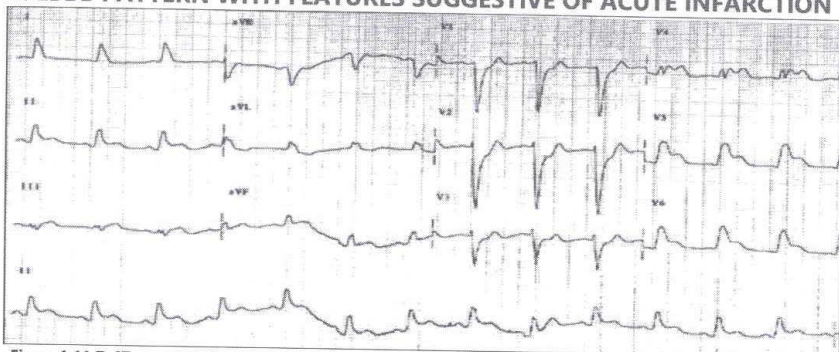
B. LBBB PATTERN WITH FEATURES SUGGESTIVE OF ACUTE INFARCTION

Figure 1.11.7. ST segment elevation >1mm in leads where the QRS complex is predominantly positive (i.e. V5, V6)

5. THE NORMAL ECG

- A normal ECG reduces the probability of AMI.
- It does not, however, reduce this probability enough to allow confident safe discharge based upon the history and ECG alone.
- Therefore, patients who present with chest pain in whom cardiac ischaemia is suspected and who have a normal ECG should undergo further diagnostic testing (i.e. delayed cardiac markers, exercise testing etc.) before they can be confidently ascribed to a low risk group.

Biochemical markers of myocardial necrosis

- Troponins are currently the preferred and recommended markers of myocardial necrosis in the setting of ACS because they are more sensitive and specific than CK-MB.
- The timing of the troponin assay is of great importance: *it needs to be delayed for 6 to 12 hours post onset of symptoms in order to safely assign a patient with a normal or non-diagnostic ECG to a low risk status.*
- In patients with an ECG suggestive of **NSTEMI or UA**, troponin assay can be performed at presentation since it will determine the diagnosis, forms part of initial risk stratification, and influences early treatment strategies.
- *Patients with ST elevation do not require urgent troponin assay since their initial treatment strategy is determined by their clinical presentation and ECG findings.*
- Troponin levels rise **within 12 hours of AMI** and remain elevated for **up to 2 weeks**: this prolonged period of elevation may mask episodes of early re-infarction.
- **Serial assays of CK-MB**, which rise and fall over a shorter time scale, may be useful in detecting re-infarction in these instances.
- Troponins can also be elevated in non-ischaemic cardiac injury or in conditions where secondary cardiac ischaemia occurs and also in renal impairment.
- **CAUSES OF TROPONIN ELEVATION OTHER THAN ACS:**
 - Myocarditis, Cardiac contusion,
 - Severe congestive cardiac failure
 - Pulmonary embolism,
 - Thoracic aortic dissection,
 - Shock of any cause
 - Renal impairment

ACS RISK STRATIFICATION**TIMI Score: Thrombolysis In Myocardial Infarction**

- The TIMI risk score can also identify patients that will benefit from certain interventions (e.g. glycoprotein IIb/IIIa (GpIIb/IIIa) Inhibitors and early percutaneous coronary intervention): the higher the risk, the greater the benefit from these interventions.
- Mnemonic "AMERICA":**
 - Age ≥ 65
 - Markers (increased serum cardiac markers)
 - ECG Changes (ST depression)
 - Risk factors (3 or more CAD risk factors: patient age (>45 M, > 55 F), family history [CAD in first degree relatives, <55 M, <65 F], hypercholesterolemia, hypertension, smoking, diabetes, obesity, sedentary lifestyle, metabolic syndrome)
 - Ischemia (2 or more anginal events over past 24 hours)
 - CAD (prior coronary stenosis of 50% or more)
 - Aspirin use within past 7 days

GRACE Score: Global Registry of Acute Coronary Events

- It is slightly less convenient to use in the ED than the TIMI score because it requires two blood assay results to complete the assessment (creatinine and troponin) and a computer to generate the risk. The predicted 6-month mortality expressed as a percentage is then stratified into a level of risk:

○ 1.5% or below: Lowest	○ >3%-6%: Intermediate	○ Over 9%: Highest
○ >1.5-3%: Low	○ >6%-9%: High	

- Treatment strategies (pharmacological and mechanical) are then recommended by NICE based on the level of risk as determined by GRACE.

HEART SCORE**HEART Score for Chest Pain Patients in the ED**

History	Highly suspicious	2 points
	Moderately suspicious	1 point
	Slightly/non-suspicious	0 point
ECG	Significant ST-depression	2 points
	Non-specific re-polarisation	1 point
	Normal	0 points
Age	≥ 65 years	2 points
	$>45-65$ years	1 point
	<45 years	0 point
Risk factors	≥ 3 risk factors or Hx CAD	2 points
	1 - 2 risk factors	1 point
	No risk factors	0 point
Troponin	$>\times 3$ normal limit	2 points
	$>1 - <3$ normal limit	1 point
	Normal limit	0 point

Risk factors: DM, Smoker, \uparrow BP, FHx of CAD, \uparrow Lipids

Score 0-3: 2.5% MACE over next 6/52 » **Discharge home**

Score 4-6: 20.3% MACE over next 6/52 » **Refer cardiology**

Score 7-10: 72.7% MACE over next 6 weeks » **Admit cardiology**

ED MANAGEMENT OF ACUTE CORONARY SYNDROMES

- **General Measures**
- Pharmacological treatment can be divided into **anti-thrombotic and anti-ischaemic**:
 - **Anti-thrombotic agents** inhibit intracoronary thrombosis through effects on the clotting cascade or via anti-platelet mechanisms.
 - **Anti-ischaemic agents** decrease myocardial oxygen demand through negative inotropic or chronotropic actions or through vasodilation.
- **MONAH**
 - **M**: Relief of pain and anxiety (**Morphine**)
 - **O**: Supplemental **oxygen** (if oxygen saturations are reduced below 94%),
 - **N**: Nitroglycerin (**GTN**)
 - **A**: Antiplatelets (**Aspirin/Clopidogrel/Ticagrelor/Prasugrel**)
 - **H**: **Heparin/Fundaparinux** (if Thrombolysis planned).

1. MANAGEMENT OF STEMI

- The priority of early therapy is to **establish reperfusion** in the affected myocardium.
- Current alternatives to achieve this goal are **Mechanical** (Primary Percutaneous Coronary Intervention (PPCI) or **Pharmacological** (Thrombolysis).

a. EARLY REPERFUSION PRIMARY PCI

- Primary Percutaneous Coronary Intervention (PPCI) is defined as angioplasty or stenting without prior or concomitant thrombolytic therapy.
- PPCI is effective in achieving and maintaining coronary artery patency without exposing the patient to the increased bleeding risks of thrombolysis.
- **PPCI is the preferred option in patients presenting with cardiogenic shock irrespective of time of onset of symptoms** and in those patients with a contra-indication to thrombolysis.

• GUIDANCE FROM NICE:

- **Offer coronary angiography, with follow-on primary PCI** if indicated, as the preferred coronary reperfusion strategy for people with acute STEMI if: presentation is **within 12 hours of onset** of symptoms and primary PCI **can be delivered within 120 minutes** of the time when fibrinolysis could have been given and in patients with **cardiogenic shock** or a **contraindication to thrombolysis**
- **Offer fibrinolysis** to people with acute STEMI presenting within 12 hours of onset of symptoms if primary PCI **cannot be delivered within 120 minutes** of the time when fibrinolysis could have been given

b. EARLY REPERFUSION THROMBOLYSIS

- There is a non-linear relationship between time delay and outcome following thrombolysis, with much greater benefit in patients with short symptom onset to treatment times: **up to 60 deaths per thousand treated are prevented if thrombolysis is delivered within one hour of onset of symptoms.**
- Bolus agents (**Retepase or Tenecteplase**) are most suitable for the pre-hospital environment. The main hazard of thrombolysis is **haemorrhage** and, in particular, **intracranial haemorrhage**.
- **Advanced age is not, in itself, a contra-indication to thrombolysis.**
- Whilst there is an increased risk of intracranial haemorrhage associated with thrombolysis in the elderly, overall mortality is significantly reduced by thrombolytic therapy in patients over the age of 75 years who present within 12 hours of onset of symptoms.

• CONTRAINDICATIONS TO THROMBOLYSIS

ABSOLUTE CONTRAINDICATIONS	RELATIVE CONTRAINDICATIONS
<ul style="list-style-type: none"> • Haemorrhagic stroke at any time • Ischaemic stroke within 6 months • Recent major surgery (within 3 weeks) • Recent major trauma / head injury (within 3 weeks) • Recent GIT bleeding (within 1 month) • Aortic dissection • Known bleeding disorder • Neurological deficit or central nervous system neoplasm 	<ul style="list-style-type: none"> • Warfarin therapy (check INR) • Pregnancy or immediate post-partum period • Transient ischaemic attack in preceding 6 months • Prolonged or traumatic resuscitation • Systolic blood pressure > 180 mmHg • Active peptic ulcer, advanced hepatic disease • Non-compressible puncture site • Infective endocarditis

- The main limitation of thrombolysis is **failure to reperfuse** (defined by lack of resolution of ST segment elevation 90 minutes after thrombolytic administration).
- This is estimated to occur in up to 30% of patients. These patients should be referred for **Rescue PCI** (PCI performed on a coronary artery that has remained occluded despite thrombolysis). Thrombolysis, even if successful at achieving early reperfusion, should not be considered the definitive treatment; **Pre-discharge angiography** (within 6-24 hours of thrombolysis) results in improved outcome and is recommended by the European Society of Cardiology: **Lyse now, stent later**.
- This should not be confused with **Facilitated PCI** which is when PCI is performed immediately after thrombolytic therapy; there is no robust evidence to support this strategy in routine clinical practice.

c. ADJUNCTIVE ANTI-THROMBOTIC THERAPY

- **Aspirin**
 - Aspirin **150 and 325 mg po**
 - Then **indefinitely at a daily dose of between 75 and 150mg**.
- **Clopidogrel**
 - Patients receiving clopidogrel, in addition to thrombolysis, aspirin and heparin, had a significantly reduced incidence of adverse events at 30 days.
- **Prasugrel**
 - *Has become the preferred option over clopidogrel in patients receiving PPCI for STEMI.*
- **Ticagrelor**
 - *Ticagrelor is being increasingly adopted instead of clopidogrel in STEMI patients undergoing PPCI who are unsuitable for prasugrel.*

Anticoagulants Rx: arterial & venous Thrombosis	MECHANISM	DRUGS	
	Direct thrombin inhibitor	<ul style="list-style-type: none"> • Dabigatran • Argatroban • Lepuridin 	
	Indirect thrombin inhibitor	<ul style="list-style-type: none"> • Heparin 	<ul style="list-style-type: none"> • Enoxaparin • UFH • LMWH
	Vit K epoxide reductase inhibitor	<ul style="list-style-type: none"> • Fondaparinux 	
	Direct Xa inhibitor	<ul style="list-style-type: none"> • Warfarin • Rivaroxaban • Apixaban 	

Antiplatelet Drugs Rx: arterial disease	Cox-1 inhibitors Glycoprotein IIb/IIIa inhibitors	<ul style="list-style-type: none"> Aspirin Abciximab Eptifibatide Tirofiban
	ADP inhibitors	<ul style="list-style-type: none"> Clopidogrel Prasugrel Ticagrelor
	Phosphodiesterase inhibitor	<ul style="list-style-type: none"> Dipyridamole Cilostazol
	Plasminogen activators	<ul style="list-style-type: none"> Streptokinase Reteplase Tenecteplase Alteplase
Thrombolytics Rx: arterial & venous Thrombosis		

2. MANAGEMENT OF NSTEMI AND UA

- A Guideline for the Initial Management of Patients with NSTEMI-ACS is shown below:

Step 1: Risk stratification using **GRACE** Risk model:

GRACE ACS Risk Model

At Admission (in-hospital/to 6 months) | At Discharge (to 6 months)

Age: Years []
HR: bpm []
SBP: mmHg []
Creat: mg/dL []
CHF: Killip Class []

☐ Cardiac arrest at admission
☐ ST-segment deviation
☐ Elevated cardiac enzymes/markers

Probability of: Death | Death or MI
In-hospital | To 6 months

SI Units | Reset | Display Score

Calculator | Instructions | GRACE Info | References | Disclaimer

Predicted 6 month mortality:

- 1.5% or below: Lowest Risk
- > 1.5-3.0%: Low Risk
- > 3.0-6.0%: Intermediate Risk
- > 6.0%: High Risk

Intervention depends on score as in table below



Step 2: Initial management of ACS as per table below:

Intervention	GRACE Risk score (predicted 6 month mortality)		
	Lowest Risk 1.5% or below	Low Risk 1.5-3%	Intermediate or High Risk >3%
	Admit AAU but refer for review cardiology within 24hrs		Admit CCU or Cardiology ward
Aspirin 300mg po stat then 75mg OD	✓	✓	✓
Bisoprolol 2.5-5mg PO OD (if no contraindication)	✓	✓	✓
Fondaparinux 2.5mg S/C OD: use UFH if eGFR <20ml/min	✓	✓	✓
Clopidogrel 600mg PO stat then 75mg daily		✓	✓
Tirofiban Infusion as per protocol and continued for 72hrs			✓
Early Angiography Within 96hrs of presentation			✓
Isosorbide dinitrate Infusion only if ongoing pain (as per protocol)			✓

III. COCAINE ASSOCIATED CHEST PAIN & AMI

PATHOPHYSIOLOGY

- **Cocaine is a powerful sympathomimetic and increases O₂ demand:**
 - Blocking re-uptake of norepinephrine and dopamine at the pre-synaptic adrenergic terminals.
 - Cocaine causes increased heart rate and blood pressure in a dose-dependent fashion.
 - By ↑ HR, BP, and contractility, cocaine leads to ↑ myocardial demand.
- **Cocaine is a potent coronary vasoconstrictor:**
 - Vasoconstriction worse with pre-existing CAD - particularly smokers.
- **Cocaine is pro-thrombotic:**
 - It increases platelet count, activation and platelet hyper-aggregability.

CLINICAL MANIFESTATIONS

- **Cardiac**
 - Coronary vasoconstriction/spasm = acute coronary syndrome
 - Exacerbated by increased myocardial oxygen demand, smoking and enhanced platelet aggregation
 - Ventricular arrhythmias
 - Hypertension with risk of aortic dissection
- **CNS**
 - Severe hypertension and focal cerebral vasospasm (enhanced by lactic acidosis, increased platelet aggregation and hyperpyrexia).
 - Cerebral infarction or haemorrhage
 - Euphoria and sense of alertness
 - Occasionally acute psychosis
 - Generalised complex epilepsy
- **Pulmonary**
 - Pulmonary oedema -? Catecholamine mediated.
 - Pneumonitis, asthma and bronchiolitis - due to immunological effects or due to adulterants in cocaine.
 - Barotrauma with smoking crack cocaine due to valsava manoeuvres thought to enhance the drug effect
- **Renal**
 - Rhabdomyolysis-induced renal failure- exacerbated by vasoconstriction
- **Obstetric**
 - Increased risk of spontaneous abortion, placental abruption
 - Intrauterine growth retardation - due to disruption of uteroplacental blood flow due to vasoconstriction and maternal hypertension

DIAGNOSIS

- **Presentation**
 - Chest pain (often "cardiac sounding") is the commonest (56%) presenting complaint amongst cocaine users.
 - Dyspnoea and shortness of breath are commonly associated.
 - Beware - up to half of cocaine associated AMIs do NOT report chest pain (palpitations or SOB etc).

- o Cocaine associated chest pain may be due to **Aortic Dissection, Pneumothorax or Pneumomediastinum**.
- **Physical**
 - o Tachycardia, Hypertension +/- Arrhythmias
 - o Tachypnoea
 - o Hyperthermia
 - o Altered mental state leading to coma +/- fits
 - o Mydriasis
 - o Diaphoresis
 - o **Consider occult trauma and associated drug use**

INVESTIGATIONS

- **Electrocardiogram**
 - o An abnormal ECG has been reported in 56% to 84% of patients with cocaine-associated chest pain.
 - o ECG sensitivity in revealing ischaemia or MI to predict a true MI is only 36%.
- **Cardiac Biomarkers**
 - o Cocaine may cause rhabdomyolysis (raised myoglobin and total CK in up to 75% of patients).
 - o Cardiac troponins are the most sensitive and specific markers.

• MANAGEMENT OF COCAINE INDUCED CHEST PAIN IN ED

- o ABCDE Approach
- o Rule out aortic dissection
- o Sedation with **benzodiazepines** which decrease central sympathetic outflow
- o Aggressive **cooling** for hyperthermia
- o Aggressive **fluid resuscitation** to maintain urine output
- o **Treat seizures with benzodiazepines** and further Rx as necessary
- o **Urgent CT brain for all seizures** as high incidence of primary intracranial pathology
- o Treat myocardial ischaemia with **Aspirin, Nitrates and/or Benzodiazepines**. **Heparin, Opiates** (MONAH).
- o Treat ventricular tachyarrhythmias and QTc prolongation with **Bicarbonate +/- Magnesium**
- o Treat severe hypertension with **Nitroprusside**.
- o **Early PCI** is particularly preferred over **fibrinolysis** in patients with cocaine-associated MI

• CONTRAINDICATED DRUGS

- o **B-blockers contraindicated** (*unopposed alpha stimulation worsens coronary and peripheral vasoconstriction*).
 - **Avoid Labetalol** as despite alpha and beta blockade the predominant effect is beta-blockade.
- o **Avoid anti-arrhythmics (including amiodarone)**
- o **Avoid Epinephrine** if cardiac arrest occurs
- o **Prolonged neuromuscular blockade** occurs with **suxamethonium** due to acquired pseudocholinesterase deficiency. Blockade rarely lasts more than 20 minutes

IV. PERICARDITIS

AETIOLOGY

- The causes of acute pericarditis are widespread and are listed in Table below.
- Most cases are **idiopathic** (80-90%). Although labelled as idiopathic, the majority of these are likely to be **viral in origin**, but viral testing is not routinely done as it rarely alters the management and is not cost-effective.

CAUSES OF ACUTE PERICARDITIS

Idiopathic 80-90%

Viral infections (echovirus, influenza virus, coxsackie B virus, HIV)

Bacterial infections (streptococci; staphylococci; gram-negative bacilli; TB⁺; in children, *Haemophilus influenzae*)

Fungal infections (histoplasmosis, coccidioidomycosis, candidiasis, blastomycosis)

Parasitic infections (toxoplasmosis, amebiasis, echinococcosis)

Autoimmune disorders (RA, SLE, systemic sclerosis)

Cancer (eg, leukemia, breast or lung cancer, and, in people with AIDS, Kaposi sarcoma)

Radiation therapy

Inflammatory disorders (amyloidosis, inflammatory bowel disease, sarcoidosis)

Uremia

Trauma

MI

Post-MI (Dressler) syndrome

Postpericardiotomy syndrome

Drugs (eg, anticoagulants, hydralazine, isoniazid, methysergide, penicillin, phenytoin,)

- The incidence of viral pericarditis is higher in young previously healthy adults and is lower in those patients who are subsequently found to need inpatient management.
- Patients with tuberculous pericarditis present with a less acute course.
- Patients with bacterial pericarditis present more acutely unwell and with other features of bacterial sepsis.
- Most cases of idiopathic pericarditis are likely to be viral in origin

CLINICAL ASSESSMENT

History

- The clinical presentation is usually one of acute onset of chest pain; classically this is pleuritic in nature and **eased by sitting up and leaning forward**.
- Pain radiating to the trapezius ridge has high degree of specificity for pericarditis

Characteristic of Pericarditis Chest pain:

- Nature:** pleuritic
- Site:** retrosternal
- Radiation:** to trapezius ridge
- Exacerbating Factors:** Worse on lying flat, eased by leaning forward,

Examination

- Pericardial friction rub:** 85% of patients
 - It is typically variable and varies with position and over time.
 - It is heard maximally during expiration
 - It is loudest at the lower left sternal edge.
 - It can be distinguished from a pleural rub by the fact that it will still be heard when the patient holds their breath.

• DIAGNOSTIC CRITERIA OF PERICARDITIS

- o A diagnosis of acute pericarditis should be made when **at least 2 out of 4** of the following criteria are met:
 - *Characteristic chest pain*
 - *Pericardial friction rub*
 - *Suggestive ECG changes*
 - *New or worsening pericardial effusion*
- o Other clinical findings associated with the aetiology of the pericarditis
 - Fever
 - Clinical features of HIV
 - Clinical features associated with autoimmune disorders
 - Patients presenting after a STEMI
 - Clinical features of Uraemia/ Metastatic Disease
- o Other clinical findings associated with the complications of pericarditis
- o **Cardiac Tamponade:**
 - The classic triad of **distended neck veins, muffled heart sounds and hypotension (Becks triad)** may not be present.
 - Patients can have an insidious onset of tamponade and the symptoms and signs may be very non-specific.
 - They may have orthopnoea, dysphagia, cough and occasionally episodes of loss of consciousness.
 - **Echocardiography** is required in all cases of suspected pericarditis in order to complete risk stratification (see below).
- o **Recurrent Pericarditis:**
 - Patients may give a history of previous resolved episodes of chest pain, or of ongoing chest pain which has required a prolonged course of NSAIDs.
- o **Chronic Pericarditis:**
 - This is defined as pericarditis lasting for more than 3 months. Symptoms include chest pain, palpitations and fatigue.

• RISK STRATIFICATION

- o The presence of any high-risk feature is associated with a poorer prognosis.
- o These patients should be admitted for inpatient management.

HIGH RISK FEATURES ASSOCIATED WITH POOR PROGNOSIS

• Temperature >38°C	• Immunosuppression	• Cardiac tamponade
• Raised WCC	• Oral anticoagulants	• Acute trauma
• Large Pericardial effusion	• Failure of NSAID therapy	• Recurrent pericarditis

• INVESTIGATION STRATEGIES

1. Electrocardiography (ECG)

- o There are a number of ECG features which are characteristic of acute pericarditis.
- o **The changes seen are diffuse; this is because pericarditis may involve a large surface area of the heart and certainly does not follow the anatomical territory of a specific coronary artery.**
 - **Widespread concave ST elevation and PR depression** throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6).
 - **Reciprocal ST depression and PR elevation** in lead aVR (\pm V1).
 - **Sinus tachycardia:** common in acute pericarditis due to pain and/or pericardial effusion.

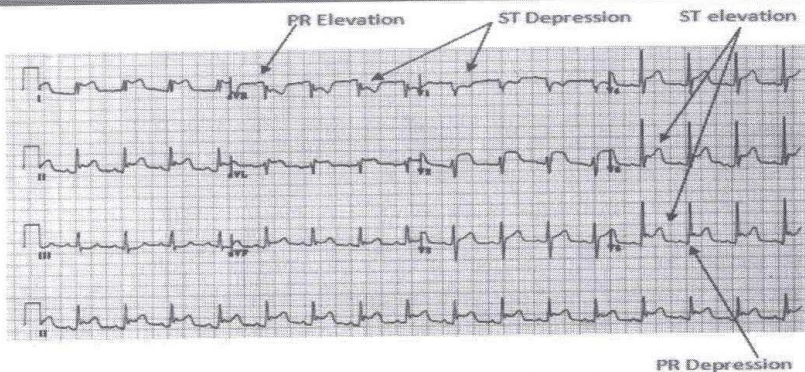


Figure 1.11.8. ECG findings characteristic of Pericarditis

PERICARDITIS vs BENIGN EARLY REPOLARISATION

- The ECG changes seen in pericarditis can be confused with Benign Early Repolarisation (BER).
- The most reliable ECG distinguishing feature is seen in lead V6. Specifically, when the **ST elevation (mm) to T wave height (mm) ratio is greater than 0.25** acute pericarditis is more likely than BER.
- ST:T wave ratio in V6 can be used to help discriminate between BER and Acute Pericarditis

PERICARDITIS	BENIGN EARLY REPOLARISATION
<ul style="list-style-type: none"> • ST segment height = 2 mm • T wave height = 4 mm • ST / T wave ratio = 0.5 • The ST / T wave ratio > 0.25 is consistent with pericarditis. 	<ul style="list-style-type: none"> • ST segment height = 1 mm • T wave height = 6 mm • ST / T wave ratio = 0.16 • The ST / T wave ratio < 0.25 is consistent with BER.

2. Troponin

- o Troponin levels may be measured and **are raised in 30-70%** of patients with acute pericarditis.
- o This does not offer any prognostic information.
- o If there is elevation in troponin it is invariably associated with **ST elevation**.
- o This creates a diagnostic dilemma and these cases may require additional specialist investigations to help make the correct diagnosis.

3. Haematology

- o A **FBC** should be performed looking for an increase in the white cell count (WCC).
- o A mild lymphocytosis is common.

- o Significantly raised WCC is an indicator of poor prognosis and will therefore make inpatient management more likely.

4. Echocardiography (ECHO)

- o The ECHO is required to evaluate for the **presence of a pericardial effusion**: if there is a **large pericardial effusion** then the patient will need to be admitted for further management.
- o Otherwise, if there are no other high risk features on evaluation, this patient can be managed at home with outpatient follow up.
- o The views obtained may be limited in obese people and those with COPD.

5. Chest X-ray (CXR)

- o CXR is generally performed to look for alternative causes of chest pain.
- o There may be radiological features of pneumonia if bacterial pericarditis is suspected or mass lesions indicative of neoplastic disease.

6. Computerised tomography of the chest (CT)

- o CT of the chest may be performed to look for alternative diagnoses such as acute aortic dissection or pulmonary embolism.

• ED MANAGEMENT OF PERICARDITIS

- o **Idiopathic**: there are no identified high risk features, pericarditis is a self limiting disease and treatment is primarily symptomatic with **NSAIDs**.
- o **NSAIDs** can be discontinued if symptoms have resolved within two weeks; if pain persists for longer.
- o If there is uncertainty about the aetiology, or there are high risk features present at initial presentation, then patients should be admitted for further investigation and management.

• DRUG TREATMENTS

DRUG	DOSE	DURATION OF THERAPY
For initial combination treatment of most patients		
Ibuprofen	400-800mg tds	1-2 weeks
Or		
Indomethacin	50mg tds	1-2 weeks
Plus		
Colchicine	0.5-0.6mg bd	3 months
For initial combination therapy patients following Myocardial Infarction		
Aspirin	650-1000mg tds	1-2 weeks
Plus		
Colchicine	0.5-0.6mg bd	3 months
For refractory cases or patients with contraindication to NSAID therapy		
Prednisolone	0.25-0.5 mg/kg/day	2 weeks
Plus		
Colchicine	0.5-0.6mg bd	3 months

COMPLICATIONS OF PERICARDITIS

- o Acute cardiac tamponade
- o Chronic constrictive pericarditis
- o Purulent pericarditis
- o Relapsing pericarditis

V. VENOUS THROMBOEMBOLISM

1. DEEP VEIN THROMBOSIS

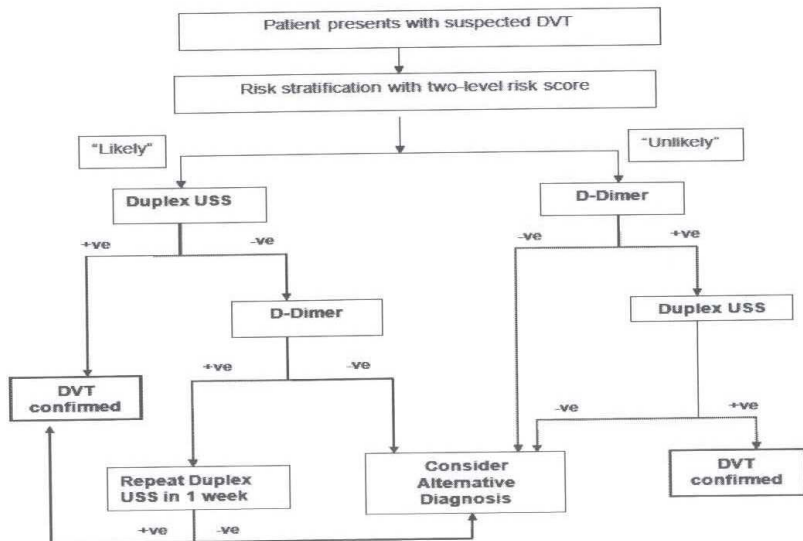
- Use Wells Clinical Model to predict pretest probability.

WELLS SCORE FOR DVT

CLINICAL FEATURES	POINTS
Active cancer (treatment ongoing, within 6 months, or palliative)	1
Paralysis, paresis or recent plaster immobilisation of the lower extremities	1
Recently bedridden for 3 days or more or major surgery within 12 weeks requiring general or regional anaesthesia	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than asymptomatic side	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Previously documented DVT	1
An alternative diagnosis is at least as likely as DVT	-2
Clinical probability simplified score	
DVT likely	2 points or more
DVT unlikely	1 point or less

DIAGNOSTIC STRATEGY PUTTING IT ALL TOGETHER

- Diagnostic approach in patients with suspected DVT is then based on above pretest probability score, use of compression ultrasound and D-dimer:



- **Pretest probability low:** perform D-dimer:
 - If negative, rules out.
 - If D-dimer positive, perform compression ultrasound (USS) proximal leg veins.
 - If USS negative – rules out.
 - If USS positive, treat.
- **Pretest probability moderate or high:** Perform USS.
 - If normal, perform D-dimer:
 - If negative, rules out.
 - If D-dimer positive, repeat USS in one week, and if still normal – rules out.
 - If abnormal, treat (and if abnormal at one week – see above).

ED MANAGEMENT OF DVT

- The mainstay of therapeutic intervention is **anticoagulation**.
- Specifically, in the UK, this is usually achieved with warfarin (oral vitamin K antagonist) in the long term, using low molecular weight heparin (**LMWH**) or **fondaparinux** as bridging therapy until the patient has achieved therapeutic anticoagulation with warfarin.

1. Pharmacological treatments:

• Bridging therapy – Heparins and Fondaparinux:

- Bridging therapy with heparin or fondaparinux should be continued for **at least 5 days** or **until the INR is 2 or greater** for at least 24 hours, whichever is longer.
- **LMWH** does not require daily monitoring and allows out-patient anticoagulation suitable for the vast majority of patients.
- **Unfractionated heparin** still has a role in patients with significant renal impairment.
- **Fondaparinux** is now licensed for the treatment of DVT when used in conjunction with warfarin.

• Warfarin:

- Warfarin treatment should be continued for a minimum of **three months**.
- Extended use beyond three months should be considered in patients with an unprovoked (ie no clear causative factor) proximal DVT if the risk of recurrence is considered high and there is no major bleeding risk.
- Patients with active cancer should receive long term (6 months) anticoagulation with LMWH (rather than warfarin): achieving therapeutic warfarin levels is difficult in cancer patients due to the increased risk of drug interactions, malnutrition, vomiting, and liver dysfunction in these patients. Moreover, cancer patients are at an increased risk of adverse effects of warfarin therapy.

• Rivaroxaban:

- This is one of the newer oral anticoagulants. It is a direct inhibitor of factor Xa; it has the advantage over warfarin in not requiring regular monitoring of the INR and not requiring a period of bridging therapy.

• Thrombolysis: Thrombolysis of venous clot is an option rarely used in the UK.

• Aspirin: Aspirin is not recommended for treatment of DVT.

2. Mechanical treatments:

- Compression stockings
- Vena caval filters

3. Other treatment issues:

- **Early ambulation** poses no risk for clot propagation and is encouraged; it may even reduce the risk of post-thrombotic complications.
- Most patients are suitable for outpatient treatment.

2. PULMONARY EMBOLISM

- Recognised clinical features found in patients with a PE are shown below:

1	Dyspnoea (70% of patients)
2	Tachypnoea (RR>20)
3	Pleuritic chest pain
4	Apprehension
5	Tachycardia (>100bpm)
6	Cough
7	Haemoptysis
8	Leg pain
9	Clinically evident DVT (10% of patients)

INVESTIGATIONS

- **ECG changes in Pulmonary Embolism:**
 - Sinus Tachycardia
 - RBBB
 - Extreme right axis deviation (+180 degrees)
 - S1 Q3 T3
 - P Pulmonale
 - T-wave inversions in V1-4 and lead III
 - Clockwise rotation with persistent S wave in V6

WELLS CRITERIA FOR P.E.

WELLS' CRITERIA	SCORE
Clinically suspected DVT	3.0
PE at least as likely or more likely than alternative diagnosis	3.0
Pulse rate >100	1.5
Immobilisation >3 days	1.5
Surgery last 4 weeks	1.5
Previous VTE	1.5
Haemoptysis	1.0
Malignancy	1.0
Clinical probability simplified scores	
PE likely	More than 4 points
PE unlikely	4 points or less

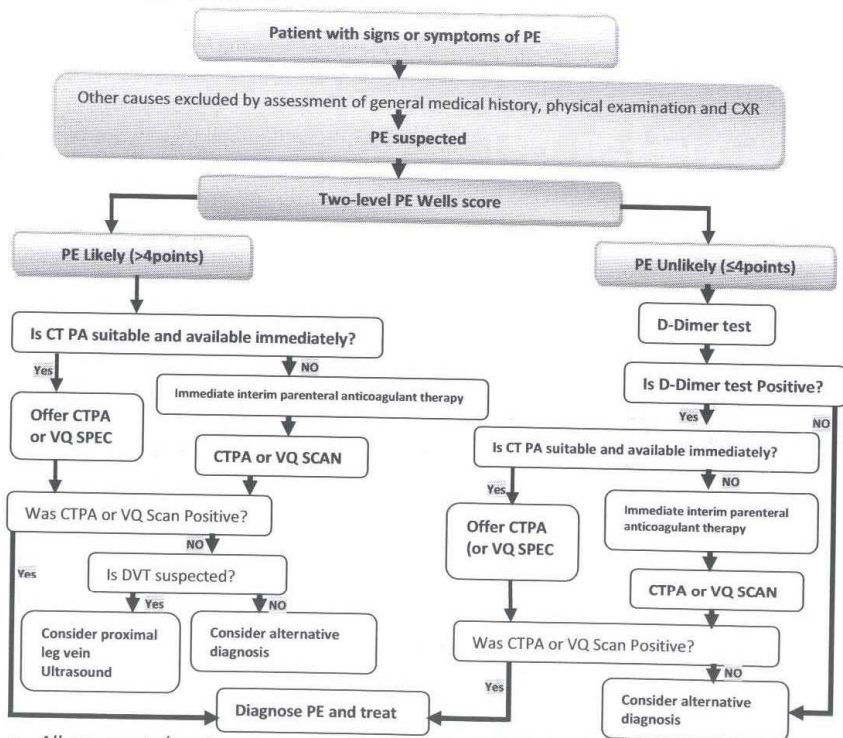
PULMONARY EMBOLUS RULE-OUT CRITERIA (PERC)

All answers to the following questions must be yes:

Low risk by Gestalt or other criteria?
Age <50?
Pulse <100?
Oxygen saturations on room air >94%?
No unilateral leg swelling?
No haemoptysis?
No recent trauma or surgery?
No previous VTE?
No oral hormone use?

DIAGNOSTIC APPROACH

- Wells' Clinical Decision Rule (CDR) to predict pretest probability – two scores with $\leq 4 =$ 'unlikely', and $> 4 =$ 'likely'.
 - If CDR score is unlikely (≤ 4), perform D-dimer:
 - If negative, rules out = no PE.
 - If D-Dimer positive, perform CTPA.
 - If CTPA negative rules out,
 - if positive, treat.
 - If CDR score likely (> 4), do CTPA.
 - If negative, rules out.
 - If positive, treat.



- All pregnant / post-partum women with suspected DVT or PE are at high risk and need definitive imaging; there is no role for a D-dimer assay.
- Imaging
 - Low and intermediate risk patients with a positive D-dimer and high risk patients require further imaging.
 - Imaging techniques include the following:
 - CT pulmonary angiogram (CTPA)
 - Isotope lung scanning (V/Q scanning)
 - Echocardiography
 - Ultrasound

- **CTPA** is the investigation of choice due to its greater sensitivity and specificity for PE than **V/Q scanning** and its ability to identify alternate diagnoses.

MANAGEMENT OF PE IN THE ED

CLINICAL MANAGEMENT

1. PATIENTS AWAITING INVESTIGATION

- All patients in the PE likely subgroup and those in the PE unlikely subgroup who have a positive D-dimer need to receive **anticoagulation** (usually with low molecular weight heparin) whilst awaiting further investigation (e.g. via CTPA). Only if CTPA is immediately available can such anticoagulation be deferred until results are available.

2. STABLE PATIENTS WITH CONFIRMED PE

- **OXYGEN**
 - Oxygen should be administered to any patient with oxygen saturations of <94% on room air (BTS oxygen guidelines, 2008).
- **ANTICOAGULATION**
 - All patients with confirmed PE require **anticoagulation**.
 - The 2012 NICE Guidelines advocate anticoagulation for **3 months for all patients in the first instance**.
 - The decision to continue beyond 3 months needs to be evaluated based on the individual's risk of recurrences compared to risk of bleeding.
 - If there have been **multiple episodes or continuing risk factors** such as malignancy **lifelong anticoagulation** should be recommended.
 - Most centres anticoagulate patients initially with low-molecular weight heparin LMWH whilst loading with warfarin.
 - The LMWH should be continued for a minimum of **5 days** and until the **INR is 2 or greater** for at least 24 hours, whichever is longer.
 - There are some groups in whom warfarin may not be appropriate such as **IV drug misusers, pregnant patients** and patients with **liver disease or cancer**. In these groups anticoagulation is usually achieved solely with **LMWH injections**.
 - **Fondaparinux**, a newer alternative to LMWH, may be considered for certain religious groups (part of the production process of LMWH uses pigs) and patients who have had previous problems with heparin such as thrombocytopenia.

3. UNSTABLE PATIENTS WITH SUSPECTED OR CONFIRMED PE

- **THROMBOLYSIS**
 - **100 mg Alteplase infusion over 2hrs (10mg given as a bolus stat)**
 - It is indicated for patients with **severe circulatory compromise** or a picture of **massive PE**.
 - Prior proof of PE is not needed if the patient is peri-arrest and thrombolysis should be administered immediately in such patients.
 - **Unfractionated heparin 80 units/kg** should be given 3 hours after thrombolysis if the patient remains alive.
- **In the setting of massive PE: only active internal bleeding or recent intracranial bleed** are absolute contraindications to thrombolysis.
- **In patients with non-massive PE:** there is no benefit from routine thrombolysis as they normally have a good prognosis.
- NICE 2012 suggests that haemodynamically stable patients should not be given thrombolysis.

TREATMENT OF PE IN SPECIAL CIRCUMSTANCES

1. PE AND ACTIVE CANCER

- o INR control is often more difficult in patients receiving chemotherapy.
- o The current recommendation (based on case series and expert opinion) is to **continue anticoagulation for life in patients with active cancer**. Patients with active cancer are at a higher overall risk of significant bleeding as a result of anticoagulation and hence **LMWH** may be a safer option than warfarin because of its shorter half-life.

2. PE AND PREGNANCY

- o Women presenting with symptoms and signs of an acute PE should have an electrocardiogram (ECG) and a chest X-ray (CXR) performed. [New 2015]
- o **In women with suspected PE who also have symptoms and signs of DVT:**
 - **Compression duplex ultrasound** should be performed.
 - If compression ultrasonography confirms the presence of DVT, no further investigation is necessary and treatment for VTE should continue. [New 2015]
- o **In women with suspected PE without symptoms and signs of DVT:**
 - Do CXR
 - **When the CXR is normal:** Only a Perfusion part of V/Q scan is preferred.
 - **When the chest X-ray is abnormal:** CTPA should be performed in preference to a V/Q scan. [New 2015]
- o Anticoagulant treatment should be continued until PE is definitively excluded.
- o Women with suspected PE should be advised that, compared with CTPA, V/Q scanning may carry a slightly increased risk of childhood cancer but is associated with a lower risk of maternal breast cancer; in both situations, the absolute risk is very small.

TREATMENT OF PE IN PREGNANCY

- **LMWH: Enoxaparin SC 1mg/kg bd.**
- **Unfractionated Heparin:** reserved for cases of massive PE (where it may be used in combination with thrombolysis). UFH is associated with osteoporosis and thrombocytopenia and is not recommended for prolonged use.
- **A temporary IVC filter** may be inserted prior to delivery as anticoagulation will need to be stopped due to the risk of haemorrhage.
- *When VTE occurs in the antepartum period, delivery should be delayed, if possible, to allow maximum time for anticoagulation rather than putting in a filter.*
- o **Oral anticoagulation** is not given during pregnancy due to a greater bleeding risk and teratogenic risks to the developing foetus.

3. PE AND IV DRUG MIS-USERS

- o The mechanism of formation of DVT in this group is probably related to repeated femoral vein puncture with injection of irritant substances, leading to endothelial damage and local release of tissue factor.
- o **Treatment of PE in Intravenous Drug Abusers**
 - Oral anticoagulants are rarely prescribed due to the risk of non-compliance with medication or monitoring. Patients who continue to inject into large veins also present a persistent bleeding risk.
 - **LMWH: Enoxaparin SC 1mg/kg bd for 3-6months**
 - **Antibiotics** given that PE in this group is often associated with sepsis
 - **IVC filters** may be useful in patients with persistent risks for DVT and PE in whom long term anticoagulation is unacceptable

CHAPTER 12. ACUTE CONFUSION / DELIRIUM**• OVERVIEW**

- Delirium is a disturbance of consciousness with inattention accompanied by a change in cognition or perceptual disturbance that develops over a short period of time and fluctuates over time (DSM4).
- **Inattention** is one of the hallmarks and pivotal features of delirium
- 3 subtypes: hyperactive, hypoactive and mixed
- Prevalence in the critically ill is about 80%

• PATHOPHYSIOLOGY

- Complex and poorly understood
- Altered cerebral blood flow
- Numerous biomarkers e.g. s100beta protein, neuron specific enolase, ILs

• RISK-FACTORS OF DELIRIUM

RISK FACTORS	PRECIPITATING FACTORS
<ul style="list-style-type: none"> • Old age • Severe illness • Dementia • Physical frailty • Admission with infection or dehydration • Visual impairment • Polypharmacy • Surgery (eg.NOF) • Alcohol excess • Renal impairment 	<ul style="list-style-type: none"> • Immobility • Use of physical restraint • Use of bladder catheter • Iatrogenic events • Malnutrition • Psychoactive medications • Intercurrent illness • Dehydration

• CAUSES OF DELIRIUM: "I WATCH DEATH"

- **I**- Infections – Pneumonia, urinary, skin/soft tissue, CNS
- **W**- Withdrawal – often unintentional, from alcohol, sedatives, barbiturates
- **A**- Acute metabolic changes – altered pH, hypo/hyper Na⁺/ Ca⁺⁺, acute liver or renal failure
- **T**- Trauma – brain injury, subdural hematoma
- **C**-CNS pathology – post-ictal, stroke, tumour, brain mets
- **H**- Hypoxia – CHF, anemia
- **D**- Deficiencies – thiamine, niacin, B12 (e.g. chronic G and T alcoholics)
- **E**-Endocrinopathies – hypo-/hyper-cortisol, hypoglycemia
- **A**-Acute vascular – hypertensive encephalopathy, septic hypotension
- **T**- Toxins and Drugs – especially anti-cholinergics, opioids, benzodiazepines
- **H**- Heavy metals

○ LIFE-THREATENING CAUSES: "WHIP X 2"

- *Wernickes, Withdrawal*
- *Hypertensive encephalopathy, Hypoglycemia and metabolic/endocrine*
- *Infection, Intracranial disease*
- *Poisons, and Porphyria*

- **ASSESSMENT**

- **CLINICAL PRESENTATION**

- **Mixed:** mixture of hyperactive and hypoactive features
 - **Hyperactive:** agitation, hypervigilance, irritability, lack of concentration, and perseveration
 - **Hypoactive:** diminished alertness, absence of or slowed speech, hypokinesia, and lethargy

- **ASSESSMENT APPROACH**

- Focussed History, Examination and Investigations
 - Assess for predisposing, Precipitating and Perpetuating factors (e.g. features of underlying illness).

- **MANAGEMENT OF DELIRIUM IN ED**

- **EARLY RECOGNITION**

- Routine monitoring
 - Seek and treat cause — especially life-threatening causes (**WHIP x 2**)

- **NON-PHARMACOLOGIC TREATMENT**

- Recurrent orientation of patients
 - Early mobilisation and physiotherapy
 - Early removal of catheters
 - Day-night routine
 - Sleep hygiene
 - Involve family
 - Noise control at night
 - Correct vision and Hearing impairment

- **PHARMACOLOGIC TREATMENT**

- *Decrease analgesics, sedatives and anticholinergic drugs, e.g. protocolised sedation or daily interrupted sedation*
 - **Thiamine** (if suspect alcohol consumption or poor nutrition)
 - **Atypical antipsychotics** (evidence suggests may reduce duration of delirium)
 - **Dexmedetomidine** (less delirium than benzodiazepine infusions, and a recent meta-analysis also suggests less than propofol infusions too)
 - **Lorazepam/ Midazolam and Haloperidol/ Droperidol** may be required for acute chemical restraint
 - NOTE there are NO FDA approved drugs for the treatment of delirium
 - No strong evidence for a pharmacological delirium protocol or any specific drugs in preventing delirium.
 - **Rivastigmine** (cholinesterase inhibitor) should not be used (increased mortality in one study)

CHAPTER 13. CYANOSIS

• DEFINITION

- **Cyanosis** is defined as a bluish discoloration, especially of the skin and mucous membranes, due to excessive concentration of deoxyhemoglobin in the blood caused by deoxygenation.
- Cyanosis is divided into two main types: **Central** (around the core, lips, and tongue) and **Peripheral** (only the extremities or fingers).

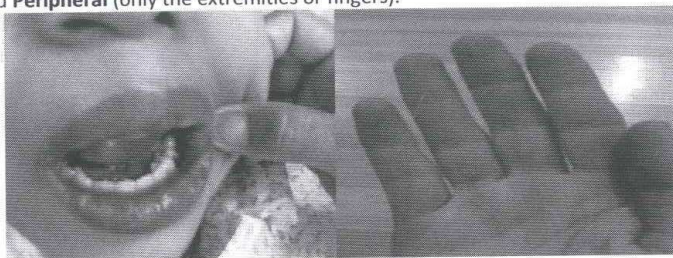


Figure 1.13.1 central and Peripheral cyanosis

I. CENTRAL CYANOSIS

- Central cyanosis is often due to a circulatory or ventilatory problem that leads to poor blood **oxygenation** in the lungs. It develops when **arterial oxygen saturation drops to $\leq 85\%$ or $\leq 75\%$** . Acute cyanosis can be a result of **asphyxiation or choking**, and is one of the surest signs that respiration is being blocked.
- Central cyanosis may be due to the following causes:
 - **Central nervous system** (impairing normal ventilation):
 - Intracranial Hemorrhage
 - Drug overdose (e.g. Heroin)
 - Tonic-clonic seizure (e.g. Grand Mal seizure)
 - **Respiratory system**
 - Pneumonia, Bronchiolitis, Bronchospasm (e.g. asthma), COPD (emphysema)
 - Pulmonary hypertension, Pulmonary embolism, Hypoventilation
 - **Cardiovascular diseases**
 - Congenital heart disease (e.g. Tetralogy of Fallot, right to left shunts in heart or great vessels)
 - Heart failure, Valvular heart disease, Myocardial infarction
 - **Blood**
 - **Methemoglobinemia** * Note this causes "**spurious**" cyanosis, in that, since methemoglobin appears blue, the patient can appear cyanosed even in the presence of a normal arterial oxygen level.
 - **Polycythaemia**
 - **Congenital cyanosis** (HbM Boston) arises from a mutation in the α -codon which results in a change of primary sequence, H \rightarrow Y. **Tyrosine** stabilises the Fe(III) form (**oxyhaemoglobin**) creating a permanent T-state of Hb.
 - **Others**
 - High altitude, cyanosis may develop in ascents to altitudes >2400 m.
 - Hypothermia, Obstructive sleep apnea

II. PERIPHERAL CYANOSIS

- Peripheral cyanosis is the blue tint in fingers or extremities, due to inadequate or obstructed circulation.
- The blood reaching the extremities is not oxygen rich and when viewed through the skin a combination of factors can lead to the appearance of a **blue color**.
- All factors contributing to central cyanosis can also cause peripheral symptoms to appear, however peripheral cyanosis can be observed in the absence of heart or lung failures.
- Small blood vessels may be restricted and can be treated by increasing the normal oxygenation level of the blood.
- Peripheral cyanosis may be due to the following causes:
 - All common causes of central cyanosis
 - Reduced cardiac output (e.g. **Heart failure, Hypovolaemia**)
 - Cold exposure
 - Arterial obstruction (e.g. **Peripheral Vascular Disease, Raynaud Phenomenon**)
 - Venous obstruction (e.g. **Deep Vein Thrombosis**)

III. METHAEMOGLOBINAEMIA

• OVERVIEW

- Methemoglobinemia is a life-threatening condition that can be congenital or acquired.
- It is characterized by the inability of hemoglobin to carry oxygen because the ferrous part of the heme molecule has been oxidized to a ferric state.
- **Acquired methemoglobinemia** is due to medications or chemicals that cause the rate of methemoglobin formation to exceed its rate of reduction. **Normal level is < 1.5%**

• CAUSES

- **Congenital:** Cytochrome B5 reductase deficiency and Haemoglobin M disease
- **Acquired (toxin/drugs):** Aniline dyes, Benzene derivatives, Chloroquine, Dapsone, Prilocaine, Metoclopramide, Nitrites (nitroglycerin, NO, sodium nitroprusside), Sulphonamides

• CLINICAL FEATURES

- Cyanosis
- Symptoms and signs of decreased oxygen delivery e.g. chest pain, dyspnea, altered mental state, end organ damage
- **SpO₂ reading 85-90%**
- Blood samples typically have a **chocolate brown hue**
- **Normal PaO₂**

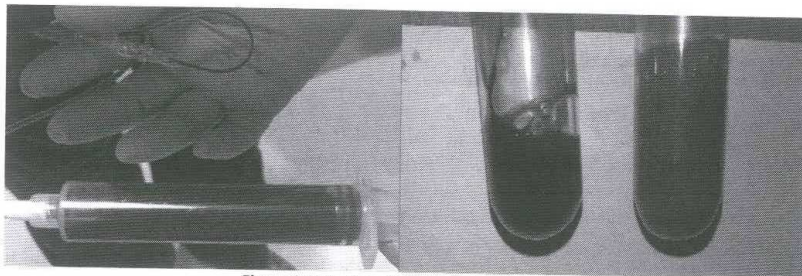


Figure 1.13.2. chocolate brown hue blood

- **DIAGNOSIS**

- The diagnosis of methemoglobinemia is based on clinical assessment when respiratory status does not explain the cyanosis that a patient has and **is refractory to oxygen therapy**.
- **Arterial blood is chocolate brown**, and the blood gas analysis indicates a **PaO₂ that is inappropriately high or normal**.
- Pulse oximetry is of little value because methemoglobin absorbs both wavelengths of light that are used in pulse oximetry.
- The definitive diagnostic test is **multiple wave length co-oximetry**.
- In patients with methemoglobinemia, PaO₂ determined by using arterial blood gas analysis **is falsely elevated**, and pulse oximetry measurements are inaccurate.
- Co-oximetry determines the true amount of oxygen saturation, which is much lower than the calculated oxygen saturation.

- **MANAGEMENT**

- **Resuscitation**

- **High flow O₂** (to ensure available Hb is saturated well)

- **Specific therapy**

- Congenital: avoid precipitants
- Cessation of precipitants
- **Methylene blue 1-2mg/kg over 5 minutes** indicated if:
 - Symptomatic
 - Consider if asymptomatic with **>20% MetHb**, or **>10% if risk factors** such as anaemia or ischemic heart disease
- Repeat methylene blue at 30-60 min if inadequate response
- Alternatives to methylene blue:
 - **Ascorbic acid** (if methylene blue contra-indicated, e.g. **G6PD deficiency**)
 - **Exchange transfusion**
 - **Hyperbaric oxygen**
- Supportive care and monitoring

- **Reasons for Failure of Methylene Blue**

- Consider the following if MetHb levels do not fall with methylene blue:
 - Massive ongoing exposure to an oxidising agent
 - Sulfhaemoglobinemia (e.g. dapson, sulfonamides)
 - G6PD deficiency
 - Methaemoglobin reductase deficiency
 - Abnormal haemoglobin
 - Excessive methylene blue (paradoxical effect in high doses)

IV. SULFHEMOGLOBIN

- Sulfhemoglobinemia is a rare condition caused by sulfur binding with hemoglobin so that oxygen cannot be bound.
- Unlike metHb, the iron moiety remains in the reduced state (HbFe⁺²).
- Sulfhemoglobin is similar to metHb in **causing low SaO₂ but not affecting PaO₂** and in imparting an intense bluish color to the skin.
- Treatment consists of **detecting the causative drug and avoiding it**.

CHAPTER 14. ACUTE DIARRHOEA IN ADULT

• OVERVIEW

- Common in ICU
- Pathophysiology: secretory, motor, exudative, osmotic

CAUSES	
○ Infective	○ Non-infective
<ul style="list-style-type: none"> ▪ Pseudomembranous colitis (Clostridium difficile) ▪ Staphylococcal enterocolitis and typhilitis ▪ Campylobacter species ▪ Clostridium perfringens ▪ Salmonella species ▪ Shigella species ▪ Escherichia coli ▪ Yersinia species ▪ Entamoeba histolytica ▪ HIV 	<ul style="list-style-type: none"> ▪ Enteral feed associated – site, mode of administration and composition ▪ Ischaemic colitis ▪ Chemical colitis ▪ Gut dysmotility ▪ Hypoalbuminaemia ▪ Acute exacerbation of Crohn's or Ulcerative Colitis
	○ Other Risk Factors
	<ul style="list-style-type: none"> ▪ Fever or hypothermia ▪ Presence of an infection ▪ Malnutrition ▪ Hypoalbuminaemia ▪ Sepsis syndrome ▪ Previous TPN

NONINFLAMMATORY vs. INFLAMMATORY DIARRHEAL SYNDROMES

FACTOR	NONINFLAMMATORY	INFLAMMATORY
Etiology	Usually viral, but can be bacterial or parasitic	Generally invasive or toxin-producing bacteria
Pathophysiology	More likely to promote intestinal secretion without significant disruption in the intestinal mucosa	More likely to disrupt mucosal integrity, which may lead to tissue invasion and destruction
History and examination findings	Nausea, vomiting; normothermia; abdominal cramping; larger stool volume; nonbloody, watery stool	Fever, abdominal pain, tenesmus, smaller stool volume, bloody stool
Laboratory findings	Absence of fecal leukocytes	Presence of fecal leukocytes
Common pathogens	<i>Enterotoxigenic Escherichia coli</i> , <i>Clostridium perfringens</i> , <i>Bacillus cereus</i> , <i>Staphylococcus aureus</i> , <i>Rotavirus</i> , <i>Norovirus</i> , <i>Giardia</i> , <i>Cryptosporidium</i> , <i>Vibrio cholerae</i>	<i>Salmonella</i> (non-Typhi species), <i>Shigella</i> , <i>Campylobacter</i> , Shiga toxin-producing <i>E. coli</i> , enteroinvasive <i>E. coli</i> , <i>Clostridium difficile</i> , <i>Entamoeba histolytica</i> , <i>Yersinia</i>
Other	Generally milder disease Severe fluid loss can still occur, especially in malnourished patients	Generally more severe disease

- **CONSEQUENCES**

- Malnutrition
- Haemodynamic instability
- Metabolic acidosis (loss of electrolytes and HCO_3^-)
- Hypokalaemia
- Hypomagnesaemia
- Low zinc
- Impaired wound healing
- Arrhythmias

- **MANAGEMENT**

- Resuscitation
- IV rehydration (Na^+ and glucose containing solutions)

- **ACID-BASE AND ELECTROLYTES**

- **Specific Therapy**

- **Primary diarrhoea:** only indicated in the really sick \rightarrow **ciprofloxacin** or **norfloxacin**
- Antibiotic induced diarrhoea: **Metronidazole**
- Can consider oral opioids or anti-diarrhoeal's: **loperamide** or **codeine**
- Enteral feeds with low osmolarity and enriched with fiber
- **Probiotics:** viable microorganisms which help host
- **Prebiotics:** non-digestible food ingredient that selects helpful bacteria in the colon
- **Synbiotics:** both probiotics and prebiotics.

- **Underlying Cause**

- Stop offending drugs/antibiotics

KEY RECOMMENDATIONS FOR PRACTICE

- In patients with acute diarrhea, **stool cultures** should be reserved for grossly bloody stool, severe dehydration, signs of inflammatory disease, symptoms lasting more than three to seven days, immunosuppression, and suspected nosocomial infections.
- Testing for *Clostridium difficile* toxins A and B should be performed in patients who develop unexplained diarrhea after three days of hospitalization.
- Routine testing for ova and parasites in acute diarrhea is not necessary in developed countries, unless the patient is in a high-risk group (i.e., diarrhea lasting more than seven days, especially if associated with infants in day care or travel to mountainous regions; diarrhea in patients with AIDS or men who have sex with men; community waterborne outbreaks; or bloody diarrhea with few fecal leukocytes).
- The first step to treating acute diarrhea is **rehydration**, preferably **oral rehydration**.
- Combination **loperamide/simethicone** may provide faster and more complete relief of acute nonspecific diarrhea and gas-related discomfort than either medication alone.
- **Antibiotics** (usually a quinolone) reduce the duration and severity of traveler's diarrhea.

I. CLOSTRIDIUM DIFFICILE

• GENERAL MANAGEMENT PRINCIPLES

- An important initial step in the treatment of *C. difficile* infection (CDI) is **cessation of the inciting antibiotic as soon as possible**.
- Treatment with concomitant antibiotics (ie, antibiotics other than those given to treat *C. difficile* infection) is associated both with significant prolongation of diarrhea and with increased risk of recurrent *C. difficile* infection.
- If ongoing antibiotics are essential for treatment of the primary infection, it may be prudent, if possible, to select antibiotic therapy that is less frequently implicated in antibiotic-associated CDI, such as parenteral **aminoglycosides**, **sulfonamides**, **macrolides**, **vancomycin**, or **tetracycline**.
- Management must also include implementation of **infection control policies**.
- Patients with suspected or proven *C. difficile* infection should **be placed on contact precautions**, and healthcare workers should **wash hands before and after patient contact**.
- **Hand hygiene with soap and water** may be more effective than alcohol-based hand sanitizers in removing *C. difficile* spores, since *C. difficile* spores are resistant to killing by alcohol. In addition, antimotility agents such as **loperamide** and **opiates** have traditionally been avoided in CDI, but the evidence that they cause harm is equivocal.
- Supportive care with attention to **correction of fluid losses and electrolyte imbalances** is also important. Patients may have regular diet as tolerated, unless surgery or other procedure is planned.

Treatment of *Clostridium difficile*-associated diarrhea in adults

- Patient with a diagnosis of CDI should have any precipitating antibiotics discontinued. If it is not possible to discontinue the precipitating antibiotics immediately then an alternative antibiotic regimen should be considered.
- All suspected cases must be isolated

Non – severe CDI	Severe CDI	Life-threatening CDI
<p>Metronidazole 400mg po tds X 10-14 days</p> <p>↓</p> <p>Daily assessment (including review of fluid/ electrolytes)</p> <p>↓</p> <p>Symptoms not improving/ worsening:</p> <ul style="list-style-type: none"> • Re-assess after 5 days, or if evidence of severe CDI (as defined below) • Switch to Vancomycin 125mg PO QID X 10-14days 	<p>Vancomycin 125 mg po QID X 10-14 days</p> <p>↓</p> <p>Daily assessment (including review of fluid/ electrolytes)</p> <p>↓</p> <p>Symptoms not improving/ worsening:</p> <ul style="list-style-type: none"> • Re-assess after 5 days, or if evidence of life-threatening CDI (as defined below) • Surgery/gastro/micro-consultation 	<p>Vancomycin 125-250 mg PO/NGT QID ± Metronidazole 500mg IV tds Consider intra-colonic vancomycin</p> <p>↓</p> <p>Consider colectomy if megacolon (caecal dilatation >10cm), perforation or septic shock</p> <p>↓</p> <p>Daily assessment (including review of fluid/ electrolytes)</p> <p>↓</p> <p>Symptoms not improving/ worsening:</p> <ul style="list-style-type: none"> • Consider high dose Vancomycin 500mg PO/NGT QID • Consider IV immunoglobulin 400mg/Kg single dose which can be repeated 21 days later if necessary

II. TRAVELERS' DIARRHEA

INFECTIOUS AGENTS

COMMON PATHOGENS IN TRAVELLER'S DIARRHOEA		
Bacterial – commonest	Viral	Parasitic
<ul style="list-style-type: none"> • <i>Escherichia coli</i>- enterotoxic or enteroinvasive – haemorrhagic • <i>Shigella</i> • <i>Campylobacter</i> • <i>Salmonella</i> • Others such as <i>Vibrio</i>, <i>Yersinia</i> 	<ul style="list-style-type: none"> • Rotavirus – children • Noroviruses – cruise ships • Astrovirus 	<ul style="list-style-type: none"> • <i>Giardia lamblia</i> • <i>Entamoeba histolytica</i> • <i>Strongyloides stercoralis</i>

OCCURRENCE

- The most important determinant of risk is travel destination, and there are regional differences in both the risk for and etiology of diarrhea. The world is generally divided into 3 grades of risk: low, intermediate, and high.
- Low-risk countries include the United States, Canada, Australia, New Zealand, Japan, and countries in Northern and Western Europe.
- Intermediate-risk countries include those in Eastern Europe, South Africa, and some of the Caribbean islands. High-risk areas include most of Asia, the Middle East, Africa, Mexico, and Central and South America.

RISK FOR TRAVELERS

- TD occurs equally in male and female travelers and is more common in young adult travelers than in older travelers.
- In short-term travelers, bouts of TD do not appear to protect against future attacks, and >1 episode of TD may occur during a single trip.
- In environments where large numbers of people do not have access to plumbing or latrines, the amount of stool contamination in the environment will be higher and more accessible to flies.
- Inadequate electrical capacity may lead to frequent blackouts or poorly functioning refrigeration, which can result in unsafe food storage and an increased risk for disease.
- Lack of safe water may lead to contaminated foods and drinks prepared with such water; inadequate water supply may lead to shortcuts in cleaning hands, surfaces, utensils, and foods such as fruits and vegetables.
- In addition, handwashing may not be a social norm and could be an extra expense, thus there may be no handwashing stations in food preparation areas. In destinations in which effective food handling courses have been provided, the risk for TD has been demonstrated to decrease.
- However, even in developed countries, pathogens such as *Shigella sonnei* have caused TD linked to handling and preparation of food in restaurants.

CLINICAL PRESENTATION

- Bacterial and viral TD presents with the sudden onset of bothersome symptoms that can range from mild cramps and urgent loose stools to severe abdominal pain, fever, vomiting, and bloody diarrhea, although with norovirus vomiting may be more prominent.

- Protozoal diarrhea, such as that caused by *Giardia intestinalis* or *E. histolytica*, generally has a more gradual onset of low-grade symptoms, with 2–5 loose stools per day.
- The incubation period of the pathogens can be a clue to the etiology of TD:
 - Bacterial and viral pathogens have an incubation period of 6–72 hours.
 - Protozoal pathogens generally have an incubation period of 1–2 weeks and rarely present in the first few weeks of travel. An exception can be *Cyclospora cayentanensis*, which can present quickly in areas of high risk.
- Untreated bacterial diarrhea lasts 3–7 days. Viral diarrhea generally lasts 2–3 days.
- Protozoal diarrhea can persist for weeks to months without treatment.

PREVENTION

- For travelers to high-risk areas, several approaches may be recommended that can reduce, but never completely eliminate, the risk for TD.
- These include instruction regarding food and beverage selection, use of agents other than antimicrobial drugs for prophylaxis, and use of prophylactic antibiotics.
- Carrying small containers of alcohol-based hand sanitizers (containing ≥60% alcohol) may make it easier for travelers to clean their hands before eating.
- Food and Beverage Selection
- Nonantimicrobial Drugs for Prophylaxis
- Prophylactic Antibiotics

TREATMENT

- Antibiotics are the principal element in the treatment of TD and are effective in cases caused by bacterial pathogens that are susceptible to the particular antibiotic prescribed. Adjunctive agents used for symptomatic control may also be recommended.

1. ANTIBIOTICS

- As bacterial causes of TD far outnumber other microbial causes, empiric treatment with an antibiotic directed at enteric bacterial pathogens remains the best therapy for TD.
- The benefit of treating TD with antibiotics has been proven in numerous studies. The effectiveness of a particular antimicrobial depends on the etiologic agent and its antibiotic sensitivity.
- Both as empiric therapy or for treatment of a specific bacterial pathogen, first-line antibiotics include fluoroquinolones, such as ciprofloxacin or levofloxacin.
- Increasing microbial resistance to the fluoroquinolones, especially among *Campylobacter* isolates, may limit their usefulness in some destinations, such as Thailand, where *Campylobacter* is prevalent.
- Increasing cases of fluoroquinolone resistance have been reported from other destinations and in other bacterial pathogens, including *Shigella* and *Salmonella*.
- A potential alternative to the fluoroquinolones in these situations is azithromycin, although enteropathogens with decreased azithromycin susceptibility have been documented in several countries.
- Rifaximin has been approved to treat TD caused by noninvasive strains of *E. coli*.
- However, since it is often difficult for travelers to distinguish between invasive and noninvasive diarrhea, and since they would have to carry a back-up drug in the event of invasive diarrhea, the overall usefulness of rifaximin as empiric self-treatment remains to be determined.

- Single-dose or 1-day therapy for TD with a fluoroquinolone is well established, both by clinical trials and clinical experience.
- The best regimen for azithromycin treatment is not yet established.

2. ANTIMOTILITY AGENTS

- Antimotility agents provide symptomatic relief and serve as useful adjuncts to antibiotic therapy in TD.
- Synthetic opiates, such as loperamide and diphenoxylate, can reduce frequency of bowel movements and enable travelers to ride on an airplane or bus while awaiting the effects of antibiotics.
- Loperamide appears to have antisecretory properties as well.
- The safety of loperamide when used along with an antibiotic has been well established, even in cases of invasive pathogens.
- Antimotility agents are not generally recommended for patients with bloody diarrhea or those who have diarrhea and fever.
- Loperamide can be used in children, and liquid formulations are available. In practice, however, these drugs are rarely given to small children (aged <6 years).

3. ORAL REHYDRATION THERAPY

- Fluids and electrolytes are lost during TD, and replenishment is important, especially in young children or adults with chronic medical illness.
- In adult travelers who are otherwise healthy, severe dehydration resulting from TD is unusual unless vomiting is prolonged.
- Nonetheless, replacement of fluid losses remains an adjunct to other therapy and helps the traveler feel better more quickly.
- Travelers should remember to use only beverages that are sealed, treated with chlorine, boiled, or are otherwise known to be purified.
- For severe fluid loss, replacement is best accomplished with oral rehydration solution (ORS), prepared from packaged oral rehydration salts, such as those provided by the World Health Organization.
- ORS is widely available at stores and pharmacies in most developing countries. ORS is prepared by adding 1 packet to the indicated volume of boiled or treated water—generally 1 liter.
- Travelers may find most ORS formulations to be relatively unpalatable, due to their saltiness. In mild cases, rehydration can be maintained with any palatable liquid (including sports drinks), although overly sweet drinks, such as sodas, can cause osmotic diarrhea if consumed in quantity.

4. TREATMENT OF TD CAUSED BY PROTOZOA

- The most common parasitic cause of TD is *Giardia intestinalis*, and treatment options include metronidazole, tinidazole, and nitazoxanide.
- Although cryptosporidiosis is usually a self-limited illness in immunocompetent people, nitazoxanide can be considered as a treatment option.
- Cyclosporiasis is treated with trimethoprim-sulfamethoxazole. Treatment of amebiasis is with metronidazole or tinidazole, followed by treatment with a luminal agent such as iodoquinol or paromomycin.

4. TD TREATMENT FOR CHILDREN

- Children who accompany their parents on trips to high-risk destinations can contract TD as well.
- There is no reason to withhold antibiotics from children who contract TD unless they have drug allergies, are taking medications that may interact with the antibiotic, or there is a strong suspicion that the illness is viral.
- In older children and teenagers, treatment recommendations for TD follow those for adults, with possible adjustments in the dose of medication.
- Among younger children, macrolides such as azithromycin are considered first-line antibiotic therapy, although some experts now use short-course fluoroquinolone therapy (despite its not being FDA-approved for this indication in children) for travelers aged <18 years.
- Rifaximin is approved for use in children aged ≥12 years.
- Infants and younger children with TD are at higher risk for developing dehydration, which is best prevented by the early initiation of oral rehydration.
- Breastfed infants should continue to nurse on demand, and bottle-fed infants can continue to drink formula.
- Older infants and children should be encouraged to eat and may consume a regular diet. Children in diapers are at risk for developing diaper rash on their buttocks in response to the liquid stool.
- Barrier creams, such as zinc oxide or petrolatum, could be applied at the onset of diarrhea to help prevent and treat rash.

5. TROPICAL SPRUE

- Tropical sprue is a syndrome of persistent TD often associated with malabsorption, steatorrhea, and folate and vitamin B12 deficiency.
- Tropical sprue is rare in short-term travelers, and recent surveys of returned travelers suggest that it is occurring less frequently worldwide.
- Although exhaustive searches to identify the etiologic agent of tropical sprue have been futile, it bears all the hallmarks of an infectious disease.
- Exclusion of other diarrheal etiologies is required for diagnosis.
- Treatment with tetracycline 250 mg 4 times daily and folate 5 mg daily for at least 6 weeks is generally successful.
- Patients with concomitant vitamin B12 deficiency may require parenteral vitamin B12.

CHAPTER 15. DIZZINESS AND VERTIGO

INTRODUCTION

- Vertigo is defined as an illusion of rotatory movement and always implies an imbalance in the vestibular system although the symptom doesn't indicate where the imbalance originates. *Dizziness is not a diagnosis and the Emergency Physician (EP) must learn to differentiate this symptom into specifically defined types.*
- Vertigo can be **physiological or pathological**.
- **Pathological vertigo** is usefully divided into two types; **central and peripheral**.
- **Central vertigo** results from dysfunction of the central connections of the vestibular apparatus including the vestibular nuclei **in the brainstem** and their connections, especially to the cerebellum. The vestibular nerve (CN VIII) is usually considered part of the peripheral vestibular system (essentially being a peripheral nerve). However, in the case of an acoustic neuroma of CN VIII, if the neuroma is large, it can compress the **cerebellopontine angle** and result in central vertigo as well.
- **Peripheral vertigo** generally refers to vertigo which arises from dysfunction of the vestibular apparatus **in the inner ear** or its connecting vestibular nerve (CN VIII). In addition to hearing, the inner ear contains the bony labyrinth where the semicircular canals and utricle/sacculle are located. These structures sense linear and angular motion, and are essential in the maintenance of balance and various vestibular reflexes.

PERIPHERAL VERTIGO	CENTRAL VERTIGO
<ul style="list-style-type: none"> • Sudden onset • More severe vertigo symptoms • Intermittent vertigo symptoms • Severe nausea and vomiting • Positional vertigo that is affected by head movement • Absence of associated focal neurology • Nystagmus away from the side of the lesion • Hearing can be impaired (Meniere's and labyrinthitis) 	<ul style="list-style-type: none"> • Gradual onset • Milder vertigo symptoms • Constant vertigo symptoms • Milder nausea and vomiting • Fixed vertigo that is not affected by head movement • New-onset headache may be present • Presence of associated focal neurology • Nystagmus towards the side of the lesion • Hearing intact

DIFFERENTIAL DIAGNOSIS OF VERTIGO

PERIPHERAL	CENTRAL
Benign Paroxysmal Positional Vertigo (BPPV)	Vertebrobasilar ischemic stroke (cerebellar, brainstem)
Labyrinthitis (viral or post-infectious)	Vertebrobasilar hemorrhagic stroke (cerebellar, brainstem)
Meniere's disease	Demyelinating (multiple sclerosis)
	Tumor – of the cerebellar-pontine angle, brainstem or cerebellum.
CN VIII tumor	Migraine (vertebrobasilar)
Perilymphatic fistula	Partial seizure
	Infection (abscess)
	Neurodegenerative disease involving brainstem and/or cerebellum
Drug-induced (e.g., aminoglycosides)	Drug induced (e.g., anticonvulsants)

CLINICAL ASSESSMENT

- **History**
- The first decision for the EP, when assessing a patient, is to **differentiate vertigo from dizziness of another type**.
 - A patient's description of the sensation of vertigo may be subjective (I feel like I am moving (or spinning)) or objective (it feels like the world is moving (or spinning)).
 - Vertigo is **not** light-headedness when moving to a standing position (**orthostatic hypotension**) or a feeling that one is about to pass out (**pre-syncope**).
- The next step is to try and **differentiate between central and peripheral vertigo**.
- In general terms, patients with central vertigo require early radiological investigation and hospital admission whereas most patients with peripheral vertigo can be safely discharged home with appropriate follow-up. The first clues in deciding whether there is a central or peripheral cause is provided by the history;
- Other factors which are useful when taking a history are:
 - Is the episode a new event or is there a history of recurrent episodes? For example, short spells of sudden onset vertigo associated with a change in head position are likely to be caused by benign positional paroxysmal vertigo (**BPPV**).
 - **Past history of vascular disease, hypertension or stroke** all increase the likelihood of a central cause for vertigo.
 - **Recent trauma or infection of the ear** makes a peripheral cause more likely.
 - **Drugs** that are associated with vertigo include **ACE inhibitors, amiodarone, aminoglycosides, beta blockers, cocaine, phenytoin, salicylates, and sildenafil**.
- **Examination**
 - Examination of a patient with vertigo must include **otoscopy**, hearing assessment and examination of the cardiovascular and neurological systems, as abnormal findings may indicate the cause of the vertigo.
 - If a reduction in hearing is found in one ear, then **Weber's and Rinne's tests** must be performed. Looking for evidence of sensorineural hearing loss.
 - Hearing loss or tinnitus almost always indicates a peripheral cause for vertigo.

NYSTAGMUS

- Spontaneous nystagmus, when present, may indicate whether vertigo has a central or peripheral origin.

	PERIPHERAL VERTIGO	CENTRAL VERTIGO
Effect of fixation	Decreases with fixation	Persists with fixation
Fatigability	Fatigues	Does not fatigue
Direction	Usually horizontal, never vertical	Any direction
Direction on movement	One direction only	Direction of nystagmus may change with direction of gaze
Duration	Resolves within 48 hours	Persists beyond 48 hours

- If spontaneous nystagmus is not present or the diagnosis is unclear, **the head impulse test** may help the EP to reach a diagnosis. The test is almost always positive in peripheral vertigo and a negative test therefore indicates a central problem. Unfortunately, it may occasionally **be positive in cerebellar stroke**.

INVESTIGATION STRATEGIES

- Laboratory investigations are extremely unlikely to be of use.
- If a central cause for vertigo is identified or suspected, imaging by either **MRI or CT** will be required to identify the cause.

TYPES OF VERTIGO

- A number of both central and peripheral causes of vertigo may present with a first episode of prolonged spontaneous vertigo some of the most common and important are described below:

1. ACUTE VESTIBULAR NEURITIS

- The most common presentation of **prolonged peripheral vertigo** seen in the ED.
- This problem typically occurs in young and middle aged adults and is postulated to be caused by a viral infection, possibly **herpes simplex**.
- It is caused by acute inflammation of the vestibular nerve and is correctly termed vestibular neuritis **not** labyrinthitis, a label which is often confusingly used and which correctly refers to a separate condition.
- The presentation is typical of peripheral vertigo:
 - *Acute onset within minutes / hours*
 - *Exacerbated by movements of the head*
 - *Accompanying severe nausea and vomiting*
 - *No other neurological deficit*
- *There is however **no disturbance of hearing** which is often otherwise found in peripheral vertigo.*

2. CEREBELLAR STROKE

- Patients with a cerebellar stroke or TIA usually have co-existing risk factors and present with very sudden onset of central vertigo and nystagmus.
- Compared to acute vestibular neuritis patients **are usually older with pre-existing risk factors** e.g. atrial fibrillation, diabetes etc.
- Onset of symptoms is hyperacute – **within a few seconds**.
- **The vertigo is central in character:**
 - *Unaffected by head position*
 - *Little systemic upset*
 - *Generally, there are co-existing neurological deficits e.g. Ataxia, depressed level of consciousness. Rarely, vertigo may be the only finding.*
- **Nystagmus is also typically central:**
 - *Horizontal, rotatory or vertical/ Bidirectional*
 - *Not suppressed by visual fixation*

3. OTOMASTOIDITIS

- Both acute and chronic ear infections may either directly infect or release toxins into the labyrinth, causing an acute labyrinthitis and peripheral vertigo.
- The patient will normally complain of fever, ear pain, headache and hearing loss.
- Otoscopy will reveal evidence of infection.

4. PAROXYSMAL VERTIGO

- For patients presenting with paroxysmal vertigo, it is useful to subdivide them into vertigo without and with hearing loss.

WITHOUT HEARING LOSS:**4.1. TRANSIENT ISCHAEMIC ATTACK (TIA)**

- Although a TIA rarely presents as isolated central vertigo, the temporary features are identical to those of cerebellar stroke.

4.2. BENIGN POSITIONAL PAROXYSMAL VERTIGO (BPPV)

- The vertigo associated with BPPV is **short lasting and characteristically related to changes in head position**.
- The vertigo is peripheral and short lasting with fatiguing nystagmus in one direction.
- Diagnosis is confirmed by the **Hallpike manoeuvre** and can be treated with the **Epley manoeuvre** which, once taught, can be effectively self administered.

HALLPIKE TEST

- Sit the patient on the bed and turn the head 45° laterally, then quickly place the patient in the supine position and extend the head 20° degrees backwards (i.e. head over the edge of the bed).
- Ensure the patient keeps their eyes open and focuses forwards (e.g. on the examiners nose). Hold the position for 30 seconds, looking for the development of nystagmus.
- Then sit the patient up and return the head to neutral, again look for nystagmus for 30 seconds. Repeat on the opposite side. **The test is positive in BPPV when the affected ear is down**

4.3. MIGRAINE

- Migraine typically presents with episodic headaches accompanied by photophobia, nausea and vomiting. Vertigo with either central or peripheral features may occur in up to 25% of patients with acute migrainous vertigo.

WITH HEARING LOSS:

4.4. MENIERES DISEASE

- The commonest cause of acute paroxysmal vertigo with hearing loss and is caused by an **increase in the pressure and volume of endolymph**.
- A patient will normally present with an initial aura of aural fullness, followed by increasing tinnitus, fluctuating hearing loss and peripheral vertigo.
- It is a clinical diagnosis and all investigations serve to rule out other diagnoses.

4.5. ACOUSTIC NEUROMA

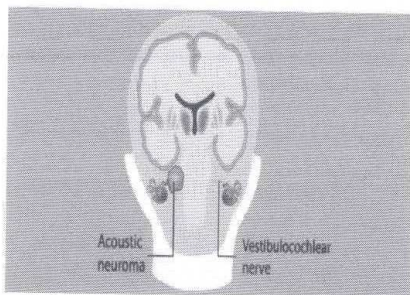
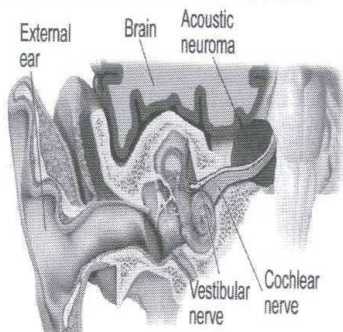


Figure 1.15.1. Acoustic Neuroma

- Acoustic neuroma is more correctly called a **vestibular schwannoma**, a benign tumour of myelin forming cells of the **vestibulocochlear nerve**.
- Unusually for a central cause of vertigo, gradual progressive hearing loss and tinnitus are common symptoms.
- Episodic central vertigo may also be a feature.

MANAGEMENT OF VERTIGO IN THE ED

- Do not expect to be able to make a definitive diagnosis in every patient with vertigo *concentrate on making a distinction between central and peripheral vertigo.*
- **In all cases of prolonged vertigo**, both the sensation of vertigo and the commonly associated nausea and vomiting, are distressing to the patient.
 - Vestibular suppressants: **Diazepam 2mg, Lorazepam 0.1mg**
 - Anticholinergics: **Hyoscine**
 - Antihistamines with anticholinergic (and antiemetic) properties: **Cyclizine.**
 - Antiemetics: **Prochlorperazine** and **Metoclopramide** are also commonly used but may be associated with **acute dystonic reactions.**
 - **Mobilisation:** Patients prescribed a vestibular suppressant should be encouraged to mobilise and to use their medication for the minimum time possible.
- **Vestibular Neuritis**
 - In addition to vestibular suppressant / antiemetic treatment, one study has shown that **steroids** may have a beneficial effect on the short and long term recovery of vestibular function although symptoms were not assessed.

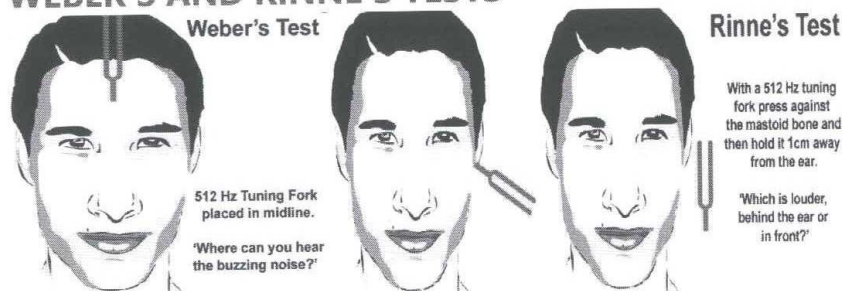
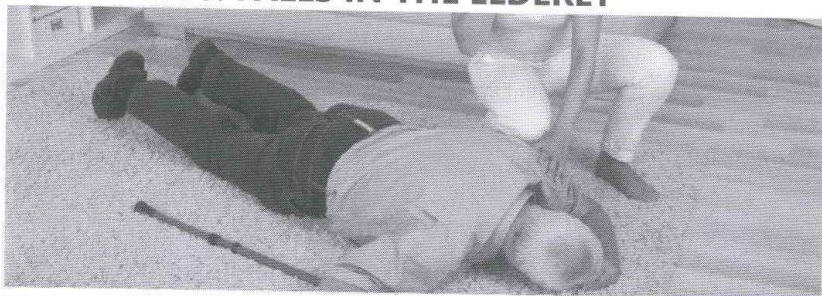
WEBER'S AND RINNE'S TESTS

Figure 1.15.2. Weber's and Rinne's tests

- **The Weber test** is done by placing a vibrating tuning fork on the patient's forehead. Either the sound will be heard equally well (or not heard!!) on both sides (which is normal) or the patient will hear the sound louder on one side. This is known as lateralising. When the sound lateralises to one ear, this means that EITHER there is a conductive hearing loss which is masking the noise on the same side, or there is sensorineural deficit on the other side.
- **The Rinne test** is done by first putting the vibrating fork close to the ear canal and then putting the fork on the mastoid region, to determine if sound is better heard at the ear canal (air conduction) or when the fork is placed on mastoid region (bone conduction). In normal circumstances, AIR conduction should always be better than BONE conduction!! If normal, then Rinne's test is POSITIVE. If bone conduction is better than air conduction then Rinne's test is NEGATIVE.
- Together these two tests will allow you to determine which ear has the hearing loss, and which type of hearing loss it is.
- If Weber's lateralises to the left, then either there is a conductive hearing loss on the left, or there is sensorineural deficit on the right.
- If Rinne's test on the left is negative, then there is conductive hearing loss on the left.
- If Rinne's test on the left is positive, then there is likely sensorineural deficit on the right.

CHAPTER 16. FALLS IN THE ELDERLY



RISK FACTORS FOR FALLS

- | | | |
|--|------------------------|------------------------|
| ○ Older age (≥ 75 years) | ○ Gait deficit | ○ Decreased hearing |
| ○ A history of previous falls | ○ Balance deficit | ○ Urinary incontinence |
| ○ Fear | ○ Visual impairment | ○ Living alone |
| ○ Acute illness | ○ Mobility impairment | ○ Home hazards |
| ○ Chronic conditions, especially neuromuscular disorders | ○ Cognitive impairment | ○ Multiple medications |

COMMON CAUSES OF FALLS IN THE ELDERLY: "I HATE FALLING"

- I** Inflammation of joints (or joint deformity)
- H** Hypotension (orthostatic blood pressure changes)
- A** Auditory and visual abnormalities
- T** Tremor (Parkinson's disease or other causes of tremor)
- E** Equilibrium (balance) problem
- F** Foot problems
- A** Arrhythmia, heart block or valvular disease
- L** Leg-length discrepancy
- L** Lack of conditioning (generalized weakness)
- I** Illness
- N** Nutrition (poor; weight loss)
- G** Gait disturbance

DRUGS THAT MAY INCREASE THE RISK OF FALLING

- Sedative-hypnotic and anxiolytic drugs (especially long-acting benzodiazepines)
- Tricyclic antidepressants
- Major tranquilizers (phenothiazines and butyrophenones)
- Antihypertensive drugs
- Cardiac medications
- Corticosteroids
- Nonsteroidal anti-inflammatory drugs
- Anticholinergic drugs
- Hypoglycemic agents
- Any medication that is likely to affect balance

IDENTIFICATION OF PEOPLE WHO HAVE FALLEN

- The recommendation from the NICE guidance on the assessment and prevention of falls in older people is that all older people in contact with healthcare professionals should be routinely asked whether they have fallen in the past year and asked about the frequency, context, and characteristics of the fall.
- Therefore, all older patients (aged 65 or older) presenting to the ED for any condition should be asked about falls.
- Older people who present to the ED following a fall or considered at risk of falling should be offered a **multifactorial risk assessment** (e.g. referred to a specialist falls service).

ASSESSMENT OF PEOPLE WHO HAVE FALLEN

- **History and examination**
- The history should ideally be obtained from the patient and, if available, a witness account of events should be sought. Pertinent points in the history include:
 - **Circumstances of events**
 - Is there a clear history of a simple trip or slip?
 - Is there history suggestive of a preceding illness (e.g. chest pain, palpitations, limb weakness, dizziness, etc.)?
 - **Loss of consciousness**
 - Is there any reported loss of consciousness or amnesia pre-or post fall?
 - Does the patient have a history of falling or collapsing?
 - Does the patient feel dizzy on sudden changes in posture?
 - **Vision:** Does the patient require glasses? When was their last eyesight test? Do they have cataracts
 - **Recent illnesses:** Has the patient had any recent illnesses that could have precipitated the 'fall' (e.g. urinary tract infection)?
 - **Past medical history**
 - Cardiac, Respiratory, Neurological, and Metabolic.
 - Have they previously sustained a fragility fracture?
 - **Drug history**
 - Are there medications that could have caused orthostatic hypotension or resulted in dizziness or poor balance (e.g. anti-hypertensives, antidepressants, antipsychotics, anticholinergics, opiates)?
 - Are there any recent medication changes?
 - **Social history:** What is the patient's social support? Do they have home hazards that are contributing to falls (e.g. loose fitting rugs, poor lighting, stairs, upstairs bathroom...)?
 - **Alcohol history:** Is there a history of alcohol excess?
- In addition to the examination of any injuries the patient should have a full cardiovascular and neurological examination to screen for evidence of any underlying cause for the fall.

INVESTIGATIONS FOR PATIENTS WHO HAVE FALLEN

- Investigations should be guided by the particular presentation of the patient and any injuries sustained. In addition, the following investigations should be considered:
 - Blood glucose/ ECG.
 - Postural blood pressures
 - Urinalysis
 - ***Creatinine kinase and renal function should be checked if the patient has had a prolonged period of immobility, to screen for rhabdomyolysis.***

ED MANAGEMENT OF A PATIENT WHO HAS FALLEN

- ED management should focus on treating the consequences of a fall and identifying any potential underlying causes.
- Patients who have fallen should be offered referral on to a specialist falls service for a multifactorial assessment.
- Prior to discharge the patient and/or carers should be given written information about the assessment they are going to receive and how to prevent further falls.

COMPLICATIONS OF FALLS

- Falling, particularly falling repeatedly, increases risk of injury, hospitalization, and death, particularly in elderly people who are frail and have preexisting disease comorbidities (eg, osteoporosis) and deficits in activities of daily living (eg, incontinence).
- Longer-term complications can include *decreased physical function, fear of falling, and institutionalization*. Falls reportedly contribute to > 40% of nursing home admissions.
- Over 50% of falls among elderly people result in an injury. Although most injuries are not serious (eg, contusions, abrasions), fall-related injuries account for about 5% of hospitalizations in patients ≥ 65 .
- About 5% of falls result in **fractures of the humerus, wrist, or pelvis**.
- About 2% of falls result in a **hip fracture**.
- Other serious injuries (**head and internal injuries, lacerations**) occur in about 10% of falls.
- Although most falls do not result in serious injury, the consequences for an individual of falling or of not being able to get up after a fall can include:
 - *Fear of further falls and thus limitation of activities. This is one of the most important effects, as unchecked it can lead to isolation, further physical decline, depression and even institutionalization*
 - *Head injury*
 - *Soft tissue injury*
 - *Fractures – wrist, hip, pelvis, rib and vertebral fractures are common*
 - About half of elderly people who fall cannot get up without help. Remaining on the floor for > 2 h after a fall increases risk of:
 - *Dehydration,*
 - *Pressure ulcers,*
 - *Rhabdomyolysis,*
 - *Hypothermia*
 - *Pneumonia.*
 - *2-5 falls may lead to hospitalisation with its own complications*

CRITICAL STEPS IN REDUCING THE RISK OF FALLS IN THE ELDERLY

- Home hazards assessment.
- Vision assessment and referral
- Strength and balance training.
- Review medication.
- Provide opportunities for socialization and encouragement.
- Improve home supports.
- Modify restraints.
- Involve the family.
- Provide follow-up.

CHAPTER 17. FEVER**I. BACTERIAL MENINGITIS****• OVERVIEW**

- Bacterial meningitis is defined as infection of the arachnoid mater, subarachnoid space, and the cerebrospinal fluid (CSF).
- Poor outcomes caused by bacterial meningitis often stem from delays in diagnosis and treatment. Initial evaluation of patients with bacterial meningitis usually occurs in ED.
- Therefore, it is critically important for ED physicians to diagnose accurately and treat promptly patients with bacterial meningitis to achieve optimal patient outcomes.

• RISK FACTORS

- CSF leak (e.g. base of skull fracture)
- Head and neck surgery or prostheses (e.g. cochlear implants, VP shunt, ICP monitor, EVD, craniectomy)
- Extremes of age (e.g. *Pneumococcus* and *listeria*)
- Head and neck infections (e.g. Sinusitis, mastoiditis, otitis media)
- Comorbidities (e.g. Liver and renal failure)
- Immunosuppression (e.g. Functional asplenia, splenectomy, hypogammaglobulinemia, complement deficiency, steroids, diabetes mellitus)
- Malnutrition/ Low socioeconomic status and overcrowding/ Exposure to epidemic

• ETIOLOGY

- **Community-Acquired Meningitis**
 - *Streptococcus pneumoniae* (**pneumococcus**) is the most common pathogen since routine immunization of infants with Haemophilus type b conjugate vaccine began.
 - However, the decrease in incidence of *H. influenzae meningitis* is seen only in vaccinated infants and children; *H. influenzae* remains among the common culprits in adult patients.
 - Along with pneumococcus and *H. influenzae*, *Neisseria meningitidis* (**meningococcus**), *Listeria monocytogenes*, and **Group B streptococci** account for nearly all of community-acquired cases in patients up to age 60.
 - Meningococcus primarily affects younger adults and is associated with individuals living in crowded spaces, such as dormitories and military barracks.
 - *Listeria* burdens persons at the extremes of age, pregnant women, and immunocompromised patients.
- **Nosocomial Meningitis**
 - Usually **Gram-negative bacilli**, especially from the **Enterobacteriaceae family**, *Staphylococcus aureus*, and **coagulase-negative staphylococci**.
 - Major risks for nosocomial meningitis include neurosurgery or head trauma within the previous month, indwelling medical devices, and CSF leak.

• CLINICAL FEATURES

- **History**
 - The classic symptoms of meningitis are **fever, stiff neck, and headache**.
 - Headaches associated with meningitis are typically nonpulsatile, nonfocal, and severe. **Altered mental status** in a patient with fever, even in the absence of headache or stiff neck, should still prompt concern for meningitis.
 - **Rash (petechial, purpuric, or even maculopapular)** in the setting of headache and stiff neck is an alarming sign of meningococcal or pneumococcal disease.

Examination

- Physical exam maneuvers traditionally have been used to evaluate neck stiffness by eliciting meningeal irritation: **Kernig and Brudzinski signs**.
- Together, these maneuvers have reportedly low sensitivity (5%) but high specificity (95%). Because of their low sensitivity and false positives among the elderly, the Kernig and Brudzinski signs have limited clinical utility.

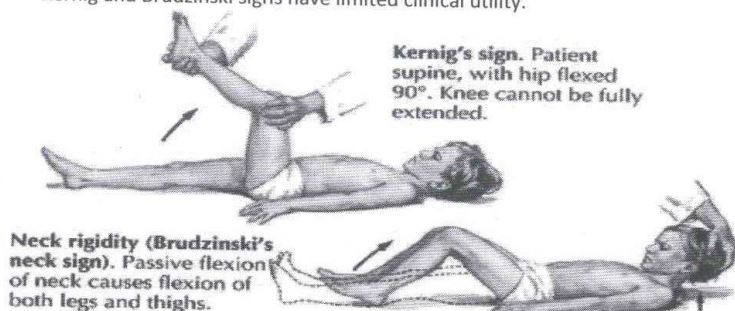


Figure 1.17.1. Kernig's and Brudzinski's signs

THE JOLT ACCENTUATION TEST

- Is an excellent maneuver to help rule out meningitis in a low-risk, nontoxic patient with headache and fever. The patient rotates his or her head horizontally at a frequency of two rotations per second; **a positive test is the exacerbation of an existing headache**.
- The jolt accentuation test has a sensitivity of 97% and specificity of 60% for the presence of CSF pleocytosis.
- Therefore, a negative test essentially can exclude meningitis in patients with fever and headache, and a positive result aids in the decision to proceed with lumbar puncture (LP).

INVESTIGATION

- LP:** Ideally prior to antibiotics
- CT Head** first if:
 - Altered mental Status
 - Focal neurologic signs
 - Papilloedema
 - Immunocompromised
 - Seizure within the previous week
- Routine blood tests, Blood cultures, Enterovirus and HSV PCR**
- Bacterial PCR** (Pneumococcus, Meningococcus), Cryptococcal antigen and India Ink
- Neurosyphilis/ Mycobacterium culture or PCR
- Immunocompromised + Gram positive rods = Listeria

Common CSF Findings in Meningitis

Index	Normal	Bacterial	Viral	Fungal
WBC/mcL	<5	>1,000	<1,000	<1,000
Differential	<15% Neutroph	>80% Neutroph	<15% Neutroph	<15% neutrophils
Glucose (mg/dL)	45-65	reduced	normal	reduced
CSF: blood glucose	0.6	reduced	normal	reduced
Protein (mg/dL)	20-45	>250	50-250	>250
Opening pressure (cm/H2O)	<20	Normal to high	Normal to high	Normal to high

ED MANAGEMENT OF MENINGITIS

- Antibiotics are essential to the treatment of bacterial meningitis. The initial choice should be governed by the patient's age and allergies, as well as resistance patterns of pathogens.
- **Vancomycin plus a third-generation cephalosporin** are the mainstays of treatment in most cases of community-acquired bacterial meningitis.
- **In patients who are older than 50 years, immunocompromised, or alcoholics, ampicillin should be added for Listerial infection.**
- Coverage for *Pseudomonas* should be added in nosocomial cases.
- In addition to antibiotics, **steroids should be given in virtually all suspected cases of bacterial meningitis.**
 - Intravenous **Dexamethasone (0.15 mg/kg)** is given just prior to or concomitantly with antibiotic administration, and continued **every 6 hours for the next 4 days.**
- Steroids have been shown to reduce overall mortality and neurological sequelae from meningitis, probably by attenuating the intense inflammatory response in the CNS.
- While this is particularly true for pneumococcus, steroids should be continued regardless of the culprit bacterial pathogen.

EMPIRIC TREATMENT**• Meningitis**

- **Ceftriaxone 2g IV BD;**
 - If Listeria risk: + **Amoxicillin 2g IV 4 hly**
 - If Strep pneumonia (pneumococcus): + **Vancomycin until sensitivity confirmed.**
 - Treat for 14 days if pneumococcus; 7 days for meningococcus
 - If severe IgE mediated reaction/anaphylaxis: **Chloramphenicol 1g IVI QID**
 - If immunocompromised: + **Vancomycin and Co-trimoxazole**

• Encephalitis

- **Acyclovir 10mg/Kg Tds** (Adjust dose in renal impairment)

• COMPLICATIONS

- **Intracranial:** Abscess, Cerebritis, Deafness, Cognitive impairment, Hydrocephalus
- **Extracranial:** Septic shock, Adrenal insufficiency from infarction (Waterhouse Friderichsen syndrome), ARF, Purpura fulminans, Necrotising vasculitis -> skin necrosis and digital gangrene.

• PUBLIC HEALTH CONSIDERATIONS

- ***Neisseria meningitidis***
 - Requires droplet precautions
 - Post-exposure prophylaxis needed for close contacts if <24h treatment with appropriate antibiotics
 - **Ciprofloxacin 500 mg** (child younger than 5 years: 30 mg/kg up to 125 mg; child 5 to 12 years: 250 mg) orally, as a single dose, OR
 - **Ceftriaxone 250 mg** (child 1 month or older: 125 mg) IM, as a single dose (preferred option for pregnant women), OR
 - **Rifampicin 600 mg** (neonate: 5 mg/kg; child: 10 mg/kg up to 600 mg) orally, 12-hourly for 2 days.

II. MALARIA

• BACKGROUND

- *Plasmodium vivax*, *ovale*, *malariae*, and *falciparum* (*P. falciparum* most virulent).
- Disease initiated by the bite of a female *Anopheles* mosquito.
- *Anopheles* mosquito originates from the tropical areas between 60° and 40°
- A careful exposure history is necessary: country and area of travel, including stopovers, and date of return. ¼ reported malaria cases in UK = *Plasmodium falciparum*.
- *P. falciparum* can lead to life-threatening multi-organ disease.
- Most non-*falciparum* malaria cases are caused by *P. vivax* (*P. ovale* or *malariae* are rare)
- Mixed infections with more than 1 species of parasite can occur.

• CLINICAL FEATURES

- *Malaria should be suspected in anyone with a fever or a history of fever who has returned from or previously visited a malaria- endemic area, regardless of whether they have taken prophylaxis.*
- Incubation period **6 days - 1 month**.
- May present more than **6 months later** (*P. vivax* and *P. ovale*).
- **No specific symptoms. Most have:**
 - Fever, headache, General malaise.
 - GI upset, jaundice or "pneumonic" symptoms uncommon.
 - Most missed malaria infections are erroneously diagnosed as non-specific viral infections, influenza, gastroenteritis or hepatitis.
 - Children are less likely to complain of chills, arthralgia/ myalgia or headaches.
 - Children may have hepato/splenomegaly.
 - Signs of hepatic de-compensation are late.

• DIAGNOSIS

- Diagnosis cannot be excluded until **3 blood specimens (thick and thin)** have been examined by an experienced microscopist.
- *P. falciparum* malaria can be diagnosed almost as accurately using **rapid diagnostic tests** (RDTs) which detect plasmodial antigens or enzymes.
- **Consider** Other travel-related infections: Typhoid, Hepatitis, Dengue, Avian influenza, SARS, HIV, Meningitis/encephalitis and Viral haemorrhagic fevers (VHF)
- **Complicated falciparum malaria**
 - **CNS:** Impaired consciousness or seizures
 - **Respiratory:** Pulmonary oedema or ARDS
 - **GIT:** Jaundice
 - **Renal:** Renal impairment
 - **Metabolic:** Acidosis (pH < 7.3), Hypoglycaemia (<2.2 mmol/l)
 - **CVS:** Shock (BP < 90/60 mmHg)
 - **Hematologic abnormalities:** Spontaneous bleeding/DIC, Anaemia (Hb <8 g/dL) and Haemoglobinuria (without G6PD deficiency/ **Black water fever**)
- Anemia in the setting of malaria occurs as a result of the following factors:
 - Hemolysis of parasitized red cells
 - Increased splenic sequestration and clearance of erythrocytes with diminished deformability
 - Shortened erythrocyte survival
 - Cytokine suppression of hematopoiesis
 - Repeated infections and ineffective treatments

• ED MANAGEMENT OF MALARIA

- **Non-falciparum malaria:**
 - 3-day course of **oral chloroquine**.
- **Dormant parasites (hypnozoites):**
 - **Primaquine**. hypnozoites persist in the liver after treatment of *P. vivax* or *P. ovale* infection: the only currently effective drug for its eradication is primaquine.
 - **Avoid primaquine in G6PD (triggers haemolysis).**
- **Uncomplicated *P. falciparum* malaria**
 - 3-day course of oral **co-artemether**: first line treatment for uncomplicated *P. falciparum*,
 - **Oral Quinine**: highly effective but poorly tolerated in prolonged dosage and is always supplemented by additional treatment, usually with **oral Doxycycline**.
 - **Atovaquone** plus **Proguanil** (Malarone).
 - **ALL** patients treated for *P. falciparum* malaria should be **admitted** to hospital for at least 24 h (may deteriorate).
- **Severe falciparum malaria, or infections with >2% of RBCs parasitized:**
 - **(Beware hypoglycaemia!!!),**
 - Should be treated with **IV Quinine**.
 - **IV artesunate** (ID consultant only) reduces high is useful in selected cases.

• BEWARE AND SEEK COMPLICATIONS:

- Some unwell patients may require haemodynamic support.
- Please seek signs of impending **ARDS, DIC, renal impairment** or **seizures**
- Severe intercurrent infections (esp. gram negative bacteraemia) may be easily missed.

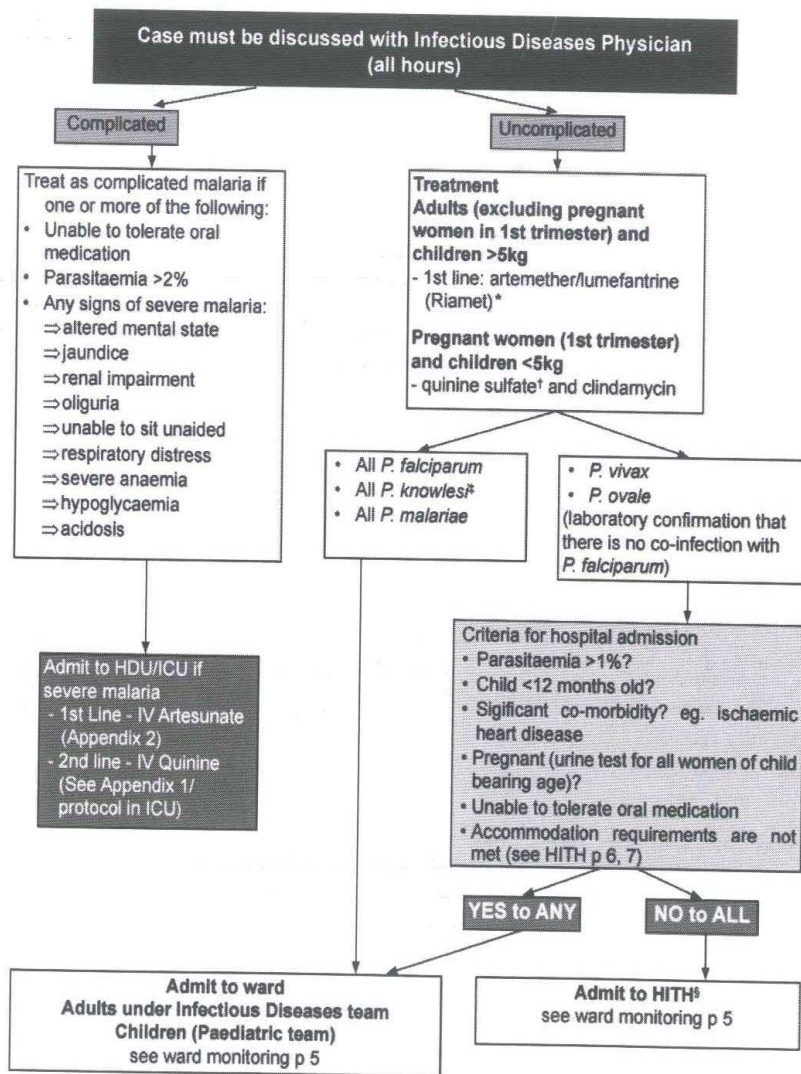
MALARIA IN PREGNANCY

- Falciparum malaria in pregnancy is more likely to be severe and complicated: the placenta contains high levels of parasites.
- The treatment of choice for falciparum malaria in pregnancy is **Quinine + Clindamycin**.
- Contraindicated in Pregnancy: **Doxycycline and Primaquine**.

MALARIA IN CHILDREN

- May present with atypical e.g. GI upset or sore throat.
- Children can be treated with most of the antimalarial regimens which are effective in adults, with appropriate dosage adjustment.
- **Doxycycline plus quinine** should not be given to children under 12 years but **clindamycin** can be substituted for doxycycline, and **pyrimethaminesulfadoxine** (Fansidar®) may also be an effective substitute.

Initial management of malaria



* Give with a small glass of milk or a soft biscuit as fat increases the absorption.

† If unable to tolerate quinine discuss with Infectious Diseases Physician.

‡ *P. knowlesi* malaria is indistinguishable from *P. malariae* on microscopy. All *P. malariae* cases with a history of travel to South East Asia should be treated as *P. knowlesi*.

§ Patients can not be discharged to Hospital in the Home (HITH) after 9pm and should be admitted at least overnight.

III. URINARY TRACT INFECTION

- **DEFINITION**

- o The term UTI encompasses a number of clinical conditions that result from the presence of microbial pathogens in the urinary tract.
- o This may involve the upper tract (kidneys and ureters), lower tract (bladder, prostate in men), or both.
- o More commonly, the terms pyelonephritis, cystitis and prostatitis are used.

- **Pyelonephritis**

- o Pyelonephritis refers to infection specifically in the renal pelvis, parenchyma and upper ureters.

- **Cystitis**

- o Cystitis refers to the inflammatory response of the bladder to infection.

- **Acute prostatitis**

- o Acute prostatitis may occur as a distinct condition, but is often associated with infection in other parts of the urinary tract.

- **Uncomplicated UTIs** occur in otherwise healthy, young, non-pregnant women with no genitourinary abnormalities.

- **Complicated UTIs** occur in certain patient populations. These include UTIs in the elderly (>65), men, in the presence of structural or functional abnormality such as obstruction and neurogenic bladder. They also include the presence of renal stones or foreign body (catheter), pregnancy, recent instrumentation or presence of comorbidity (diabetes, malignant disease).

- **Etiologies**

- o **Escherichia coli:** 75-95%
- o **Staphylococcus saprophyticus:** 5-15% (Honeymooner cystitis),
- o **Enterococci, Klebsiella and Proteus:** 5-10%.

- **E. coli** remains the dominant pathogen in complicated UTIs.

CLINICAL ASSESSMENT OF COMPLICATED UTIS

- **Cystitis**

- o Cystitis commonly presents with one or more of dysuria, urinary frequency, haematuria, urgency and suprapubic discomfort, especially in the young adult woman.

- **Pyelonephritis**

- o Pyelonephritis is characterised by fever, flank pain or tenderness with or without the symptoms of lower urinary tract infection.
- o Studies reveal younger patients lacking a fever (defined as less than 37.8 C) to often have an alternative diagnosis such as **PID, cholecystitis or renal colic**.
- o Unusual presentations of pyelonephritis are often seen with pain in the epigastric area or either hypochondrium. The vast majority of patients with UTI will be systemically well.
- o **The elderly** merit special mention. They often have complicated UTIs and the symptoms and signs are often less well localised. They may be afebrile or have only a low-grade fever. Verbalisation of their symptoms may be difficult because of acute confusion, as well as from existing medical conditions.
- o The diagnosis should be considered in the elderly presenting with reduced level of consciousness, lethargy and generalised weakness.

- o The differentiation between cystitis and pyelonephritis is important in terms of resulting morbidity and choice of antibiotic and length of treatment.
- o **Pyelonephritis** will most often require a **7-10-day course of antibiotics**. It also always requires a **renal ultrasound** to be performed. This may be acutely on admission or as part of discharge follow up.
- o The most common indications for admission are *nausea and vomiting, comorbidity (especially pregnancy) and obviously severe sepsis or shock*.
- **INVESTIGATIONS**
 - o Urine dipstick, Urine microscopy and culture
 - o Imaging: mainly ultrasound, but occasionally CT in certain complicated UTIs.
- **Imaging**
 - o It may reveal complications of urinary tract infection such as **renal calculi, hydronephrosis and renal abscess**. Severely unwell patients, those who fail to resolve and those in which diagnostic uncertainty exists, require urgent imaging.
 - o **CT** will detect any renal calculi, hydronephrosis and abscess, yet is most usually saved for renal colic or diagnostic uncertainty.

MANAGEMENT OF UTIs IN THE ED

1. ACUTE CYSTITIS

A. ACUTE UNCOMPLICATED CYSTITIS

- o In young female, non-pregnant patients in areas with low *E. coli* resistance, **trimethoprim** is still a reliable empiric treatment.
- o **Nitrofurantoin** must not be used if pyelonephritis is suspected, as it has poor efficacy in the upper urinary tract.

B. ACUTE COMPLICATED CYSTITIS

- o **Ciprofloxacin** or **cephalexin** may be used.
- o Avoid trimethoprim.

2. ACUTE PYELONEPHRITIS

A. ACUTE UNCOMPLICATED PYELONEPHRITIS

- o **Ciprofloxacin** is the initial treatment of choice for uncomplicated pyelonephritis.
- o If intravenous treatment is required, a **single dose of gentamicin** followed by ciprofloxacin is a reasonable approach.
- o The IV dose can be given in the ED allowing the patient to be discharged on oral antibiotics.
- o *Uncomplicated pyelonephritis in a well patient can usually managed as an out-patient initially.*

B. ACUTE COMPLICATED PYELONEPHRITIS

- o **Admit/ IV Ciprofloxacin, Piperacillin-Tazobactam or Imi/Meropenem.**
- o **Gentamicin** may be useful.

3. UTI IN PREGNANCY

- o UTIs in pregnancy are complicated.
- o **Cystitis:** Use **Nitrofurantoin, Cephalexin** or **Amoxicillin**.
- o **In pyelonephritis,**
 - **Ceftriaxone** may be used, but in later pregnancy there is a risk of kernicterus.
 - **Piperacillin-tazobactam** may also be used.

CHAPTER 18. FITS / SEIZURE**I. SEIZURES****OVERVIEW**

- Seizures are the manifestation of abnormal hyperexcitable discharges of cortical neurons.
- 10 minutes of continuous seizure activity is now considered status epilepticus, which may result in neuronal damage.
- Seizures should be aggressively treated if they last longer than 5 minutes.
- International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE) in 2005 definition of **Epilepsy**: brain disorder characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychologic, and social consequences of this condition.
- Traditionally, the diagnosis of epilepsy requires the occurrence of **at least 2 unprovoked seizures 24 hours apart**.

CAUSES OF SEIZURES

- **Epilepsy**
 - May be the first presentation
 - Noncompliance with anticonvulsant therapy
 - May be **triggered by specific factors**, e.g. Stress sleep deprivation, medications (e.g. tramadol, antipsychotics that lower the seizure threshold), intercurrent illness
- **Other conditions** which cause generalized or focal CNS injury can also precipitate or perpetuate seizures:
 - Hypoxia
 - Metabolic disturbances (e.g. hypoglycaemia, uremia, hepatic encephalopathy)
 - Electrolyte imbalance (e.g. hyponatraemia, hypernatraemia, hypercalcaemia)
 - Drug intoxication (e.g. anticonvulsants, antidepressants, antipsychotics, isoniazid, opioids, theophylline, sympathomimetics)
 - Drug withdrawal (e.g. alcohol, barbiturates, benzodiazepines)
 - Trauma
 - CNS neoplasia/ Stroke
 - Intracranial haemorrhage (e.g. subarachnoid hemorrhage, intracerebral hemorrhage)
 - CNS infection (e.g. meningoencephalitis, cerebral abscess, neurocysticercosis)

CAUSES OF EPILEPSY

- **Symptomatic epilepsy**: due to a known cause, e.g. head injury.
- **Idiopathic epilepsy**: where there is no apparent cause.
- **Cryptogenic epilepsy**: where there is no apparent cause but there is evidence that it may be the result of brain damage due to other associated conditions such as:
 - *Developmental condition, e.g. Autistic spectrum disorder.*
 - *Learning difficulties.*
 - *Unusual electroencephalogram (EEG) readings.*

CAUSES OF SYMPTOMATIC EPILEPSY

- Conditions that affect the structure of the brain, e.g. cerebral palsy.
- Drug and alcohol misuse.
- Birth defects, Hypoxia at birth.
- CNS infections, e.g. Meningitis.
- Head injuries, Strokes.
- Brain tumours.

TYPES OF SEIZURES

- Seizures are divided into two main types: **Partial seizures** and **Generalized seizures**.

1. PARTIAL SEIZURES

- Partial seizures originate from a specific area of the cortex; they are sometimes called '**focal**' seizures because the seizure affects just one area or 'focus'.
- There are two types of partial seizure:

A. SIMPLE PARTIAL SEIZURES

- Involve only one area of the brain and **consciousness is maintained**
- The clinical features of a simple partial seizure depend on the region of the brain affected.

B. COMPLEX PARTIAL SEIZURES

- Arise from a single region of the brain and are associated with a degree of **consciousness impairment**.
- Complex partial seizures affect a larger part of one hemisphere than a simple partial seizure. The patient's conscious level is reduced and they may be confused and have no memory of the seizure.
- Patients may be able to hear during a seizure but will often not fully understand what is being said.
- Complex partial seizures often happen in the **temporal lobes** ('temporal lobe epilepsy') but can affect other areas of the brain.

C. SECONDARY GENERALIZATION

- Partial seizures sometimes spread from one hemisphere to both hemispheres.
- This is called a secondary generalized seizure because it starts as a partial seizure and then becomes generalized.
- When this happens, the patient loses consciousness and will usually have a tonic-clonic seizure. Sometimes the partial seizure is so brief that it is not noticed.

2. GENERALIZED SEIZURES

- Generalized seizures **affect both sides of the brain at once and can happen without warning**. The patient is unconscious (except in myoclonic seizures).
- Following the seizure, the patient has no recollection of what happened during the attack.
- There are six main types of generalized seizure:

A. ABSENCE SEIZURES (PETIT MAL)

- An **absence seizure** (formerly classified as *petit mal*), is a very uncommon seizure that begins suddenly and occurs without any warning signs.
- People experiencing absence seizures typically appear to stare without moving.
- Usually lasting **less than 15 seconds**, absence seizures can occur many times a day and may be **mistaken for daydreaming**.
- While the patient may not remember, what happened during the seizure, they'll typically return to being instantly alert as soon as the seizure is finished.

B. MYOCLONIC SEIZURES

- Myoclonic seizures** (*myo* meaning "muscle", *clonic* meaning "jerk") result in an increase in muscle tone.
- The areas of the brain stem responsible for increasing and decreasing muscle tone are close together – meaning atonic and myoclonic seizures probably begin in the same place.
- Patients experiencing sudden dramatic changes in muscle tone will often suffer from either or both types of seizures.

- Patients suffering from myoclonic seizures typically react as if hit by a single jolt of electricity.
- Myoclonic seizures are often only one manifestation of a mixed seizure disorder, and they can be very serious.
- On rare occasions, a patient may suffer from a series of such jolts – a serious type of epilepsy classified as **infantile spasms**.
- It is important to note that not all myoclonic jerks are myoclonic seizures.

C. TONIC SEIZURES

- During a tonic seizure, the person's **muscles initially stiffen** and they **lose consciousness**.
- The person's **eyes roll back** into their head as the muscles (including those in the chest, arms and legs) contract and the back arches.
- As the chest muscles tighten, it becomes harder for the person to breathe – **the lips and face may take on a bluish hue**, and the person may begin to make gargling noises.
- Many observers have the misconception that the person is in danger of "swallowing their tongue," so they attempt to put something in the person's mouth.

D. CLONIC SEIZURES

- During a **clonic seizure**, the individual's **muscles begin to spasm and jerk**. The elbows, legs and head will flex, and then relax rapidly at first, but the frequency of the spasms will gradually subside until they cease altogether.
- As the jerking stops, it is common for the person to let out a deep sigh, after which normal breathing resumes.

E. TONIC-CLONIC (GRAND MAL) SEIZURES

- A tonic seizure is typically accompanied by a clonic seizure – it is rare to experience one without the other. When both are experienced at the same time, this is known as a **tonic-clonic seizure** (formerly known as a **grand mal seizure**).

F. ATONIC SEIZURES (DROP ATTACKS)

- Unlike **myoclonic seizures** (which cause muscles to tense), **atonic seizures** cause the **muscles to go limp**. The patient's body may slump or crumple to the ground, possibly causing injury.
- The areas of the brain stem responsible for increasing and decreasing muscle tone are close together, which means atonic and myoclonic seizures probably begin in the same place.
- Patients experiencing sudden dramatic changes in muscle tone will often suffer from either or both types of seizures.
- Atonic seizures may be indicative of **Lennox-Gastaut Syndrome**.

TRIGGERS FOR EPILEPTIC SEIZURES

- These triggers include:
 - Stress.
 - Lack of sleep.
 - Alcohol, particularly binge drinking and during a hangover.
 - Illegal drugs, such as cocaine, amphetamines, ecstasy, and any opiate-based drugs, such as heroin, methadone, or codeine.
 - Concurrent infections that cause a fever.
 - Flashing lights (this is an uncommon trigger that affects only 5% of people with epilepsy, and is also known as photosensitive epilepsy).
 - Menstruation.

ED APPROACH TO PATIENTS WITH SEIZURE

- Patients frequently present to the ED following a first seizure.
- Patients should not be labelled with the diagnosis of epilepsy in the ED.
- The main role of the ED is to screen patients for potential reversible causes and refer on for specialist assessment.

CLINICAL FEATURES OF A 'FIRST SEIZURE'

- A detailed history is crucial for the diagnosis and this should be obtained from the patient and any available witnesses.
- No one feature enables the diagnosis of an epileptic seizure but certain clinical features may suggest a particular diagnosis.

CLINICAL FEATURES DIFFERENTIATING SEIZURES			
Epileptic seizure	(Pseudo)/Non-epileptic seizure	Vasovagal episode	Cardiac disorders
Bitten tongue. Head-turning to one side. No memory of abnormal behaviour. Unusual posturing. Prolonged limb-jerking. Confusion after event. Prodromal <i>déjà vu</i> or <i>jamais vu</i> . Incontinence.	Poorly controlled thrashing. Back arching. Eyes held shut. Head rolling. Pelvic thrusting.	Posture —prolonged standing precipitates an attack or similar episodes have been prevented by lying down. Provoking factors , e.g. pain, medical procedure. Prodromal symptoms , e.g. sweating, feeling hot. May have short-lived, irregular myoclonic jerking. Full, rapid recovery.	Short-lived, irregular myoclonic jerking. Palpitations. Chest pain. Shortness of breath. May occur during exercise. Reduced exercise tolerance or fatigue.

- Patients should have a cardiovascular, respiratory, and neurological examination as part of their assessment in the ED.
- Examination findings that may suggest an underlying precipitant include:
 - **Fever:** suggestive of an infective process such as meningitis.
 - **Purpuric rash:** suggestive of meningococcal disease.
 - **External signs of head trauma:** which may be indicative of intracranial bleeding, e.g. subdural haematoma.
 - **Retinal haemorrhages:** in keeping with a diagnosis of subarachnoid haemorrhage.
 - **Jaundice, spider naevi,** or other signs suggestive of chronic alcohol abuse.
 - **Focal limb or facial weakness:** supportive of a diagnosis of thromboembolic stroke.
- **INVESTIGATIONS FOLLOWING A FIRST SEIZURE**
 - **Blood glucose:** to exclude hypoglycaemia as a cause.
 - **ECG:** to screen for an underlying arrhythmia, e.g. abnormal QT interval.
 - **FBC:** to screen for a potential underlying infective cause.
 - **U&E, Magnesium and Calcium:** to screen for potential metabolic causes.

- If the patient is pyrexial, then further investigation may be appropriate to identify the infective cause:
 - **Urinalysis.**
 - **CXR.**
 - **Blood cultures.**
- A CT head is not routinely required in patients following a first fit but should be performed in those where an underlying focal cause is likely, e.g. focal neurological signs, head injury, suspected intracranial infection, bleeding disorder (including anti-coagulants), or where full consciousness is not regained.

FOLLOW-UP AND DISCHARGE ADVICE

- The NICE guidance on the management of epilepsy recommends that all patients having a first fit should be seen as soon as possible (**within 2 weeks**) by a specialist in the management of epilepsy.
- Upon discharge from the ED the patient and relatives should be given the following advice (verbally and written):
 - How to **recognize** a seizure.
 - First-aid advice on how to **manage** a subsequent seizure.
 - **Safety advice**, e.g. not locking bathroom doors, swimming unsupervised, operation of heavy machinery, etc.
 - **Driving advice**: the patient must be informed they cannot drive and they must inform the DVLA. They must refrain from driving for **six months from the date of the seizure**. A subsequent diagnosis of epilepsy extends this to **1 year**.
 - Appointment with an epilepsy specialist and contact details for the service.

II. STATUS EPILEPTICUS (SE)

DEFINITIONS

- SE is a prolonged seizure of any type.
- Traditional definitions of SE refer to either:
 - A single seizure persisting for greater than 30 minutes
 - Multiple seizures of shorter duration without a full neurological recovery in between seizures
- *There is a growing body of support for the definition to refer to seizures that persist for greater than 5 minutes without intervention.*
- **Impending Status Epilepticus** has been advocated to describe continuous or intermittent seizures that persist **beyond 5 minutes** without neurological recovery.
- **Established SE** refers to clinical or electrographic seizures that persist for **30 minutes** or longer without full neurological recovery in between.
- **Refractory SE** is defined as the persistence of convulsions despite adequate doses of two intravenous agents. SE may be subdivided into convulsive and non-convulsive forms.

MANAGEMENT OF S.E. IN THE ED

- **Pre-hospital**
 - **ABC:** attention to airway, breathing and circulation, with the application of high flow oxygen where available.
 - **Blood glucose** should be checked and intravenous dextrose used to treat hypoglycaemia as indicated.
 - **Benzodiazepines:** **Diazepam** (rectal) or **Midazolam** (buccal or intranasal) may be used for this purpose.
- **On arrival in the ED**
 - Check **ABC**
 - Administer high-flow oxygen
 - Measure **blood glucose** and do **Pregnancy test**
 - **Drug regime**
 - **IV access:** Lorazepam **0.1 mg/kg IV**
 - **No IV access:** Diazepam **0.5 mg/kg PR**
- **10min later; continued seizure:**
 - **Drug regime**
 - **IV access:** Lorazepam **0.1 mg/kg IV**
 - **No IV access:** Paraldehyde **0.4 ml/kg (in same volume of olive oil) PR**
- **20min later; continued seizure:**
 - Request senior help, if not already present
 - Consider intraosseous access, consider IV cutdown if IV access not already established
 - **Drug regime**
 - Phenytoin **18 mg/kg IV** OR Phenobarbitone **20 mg/kg IV**
 - And Paraldehyde **0.4 ml/kg (in same volume of olive oil) PR** if not already given.
- **40 min later; continued seizure:**
 - Rapid sequence intubation
 - Transfer to intensive therapy unit (ITU)
 - **Drug regime:** Thiopental **4 mg/kg**

CHAPTER 19. HAEMATEMESIS & MELAENA**I. UPPER GASTROINTESTINAL HAEMORRHAGE****INTRODUCTION**

- Upper GI haemorrhage can be defined as **any bleeding which occurs from a source proximal to the Ligament of Trietz.**
- The Ligament of Trietz is the suspensory ligament of the duodenum that marks the duodeno-jejunal junction.
- In peptic ulcer disease, haemorrhage occurs from erosion to an artery at the ulcer base.
- In patients with liver disease, increased portal venous pressure results in greater blood flow through collateral vessels.

CLINICAL ASSESSMENT

- **Identify fact that patient is having an upper GI bleed**
 - Easy features Haematemesis (present in 50%) and melaena (present in 70%)
 - Not so easy syncope, dizziness, fresh PR bleeding, hypotension, tachycardia
 - Patients with haematemesis tend to have more severe bleeds than those with only melaena.
- **Points to look for in the history**
 - Known or suspected liver disease
 - Profuse recent vomiting (suggests Mallory Weiss tear)
 - Previous peptic ulcer disease or gastritis
 - Known or previous *H. pylori* infection
 - Alcohol (not direct pointer to varices as bleeding from peptic ulcer disease still more common in this group)
 - Medication particularly NSAIDs, aspirin or warfarin (implicated in approximately 20%)
 - *Do a PR early; as it may confirm fresh melaena.*
- **Identify and resuscitate patients with haemodynamic instability**
 - If there is any evidence of haemodynamic instability then involve senior ED physician.
 - **A & B: High concentration oxygen** delivered via a mask with reservoir bag
 - **Circulation/ Cannulation:**
 - **Two large bore peripheral intravenous cannulae**
 - **Bloods** (see investigations)
 - **Intravenous fluids crystalloid** (colloids if known liver disease*) administer **1-2 litres** immediately and reassess
 - If not improving administer **red cells** (O-neg if necessary)
 - **Gastric tube** and aspirate stomach widely used in US not in UK.
 - **Urinary catheter** and measure urine volumes
 - **Disposal: Urgent referral** to senior GI specialist and Critical Care
- **Perform a risk assessment**
 - **Variceal Bleeds**
 - Variceal bleeds are **uncommon** even in patients with known alcohol related liver disease.
 - Mortality is high and up to 30% of patients with known varices are likely to re-bleed.
 - A variceal bleed is suggested by evidence of decompensated liver disease such as **jaundice, ascites or encephalopathy.**

- Known or suspected variceal bleeds should always be considered to be high risk as in hospital mortality is approximately 50%.
- All patients should be referred for urgent endoscopy and admitted to a critical care area.
- Prognosis is principally related to the degree of underlying liver disease rather than the extent of bleeding.
- **Non-variceal bleeds**
 - The most widely used risk assessment tool is the **Rockall score**.

ROCKALL SCORE.

- It was found to **predict mortality but not the rate of re-bleeding**.
- The score consists of three clinical parameters (**age, presence of shock and co-morbidity**) and two parameters that rely on endoscopic findings (**blood and diagnosis**).
- The maximum pre-endoscopy Rockall score is 7 and post-endoscopy 11.
- A Rockall score of 3 before endoscopy approximates with a 10% mortality rate and a score of 6 a 50% mortality rate.
- The main disadvantage of the Rockall score is that it requires findings at endoscopy to calculate all the components of the score.

BLATCHFORD SCORE

- The **Blatchford score** was designed to **predict the need for subsequent treatment**.
- This score is more complex than the Rockall although does not include findings at endoscopy
- *Both the Blatchford and Rockall scores are useful tools in identifying high risk upper GI bleeds. Rockall scores are more widely used in the UK.*
- **Risk is categorised in the following way:**
 - **Score 0: Very low risk:** no objective evidence of GI bleed consider discharge
 - **Low risk:** admit to MAU or observation unit for next day or out-patient endoscopy
 - **Moderate risk:** admit to appropriate inpatient specialty (local protocols) for urgent endoscopy.
 - **Score ≥ 6 : High risk:** (Rockall Score (**pre-endoscopy**) $\Rightarrow 3$): haemodynamic instability, known varices resuscitate, admit to critical care area for emergency endoscopy.
- **DIFFERENTIAL DIAGNOSIS**
 - *Peptic ulcer disease is the commonest cause of upper GI bleeding*
 - *Portal hypertensive gastropathy*
 - *Gastric or duodenal varices*
 - *Aorto-enteric fistula*
- **INVESTIGATION STRATEGIES**
 - 12-lead ECG
 - ABG, FBC, U&E, LFT, Coagulation Screen, Cross Match RCC
 - PR and FOB, if no melaena or unsure
 - CXR
 - Little role for abdominal x-ray

MANAGEMENT OF UGIT BLEEDS

- **Medical therapy (non-variceal bleeds)**
 - **High dose IV PPIs:** Pantoprazole 40mg IV.
 - **Somatostatins**
 - **Octreotide Infusions** are used occasionally in severe acute non-variceal upper GI haemorrhage.
 - **Antifibrinolytic therapy**
 - **Tranexamic Acid:** There is little evidence to support this.

VARICEAL HAEMORRHAGE**MANAGEMENT OF VARICEAL BLEEDS**

Treatment of Acute Variceal Hemorrhage	
Regimen	Options
General Management	Admit to intensive care unit
	Resuscitation but limit transfusion to hemoglobin level of 7 g/dL
	Secure intravenous access
	Consider intubation and mechanical ventilation
Vasoconstrictor	IV octreotide (50 µg bolus then 50 µg/hour infusion x 2 to 5 days)
	IV terlipressin (2 mg every 4 hours for first 48 hours, followed by 1 mg every 4 hours x 2 to 5 days)
Antibiotic Prophylaxis	IV ceftriaxone 1 gm daily x 7 days (preferred in Child class B and C)
	Oral norfloxacin 400 mg twice a day x 7 days
Endoscopic Therapy	Endoscopic variceal ligation (preferred)
	Endoscopic variceal sclerotherapy
Salvage Therapy	Balloon tamponade (only temporary, maximum 24 hours)
	Transjugular intrahepatic portosystemic shunt

BALLOON TAMPONADE (SENGSTAKEN-BLAKEMORE TUBE):

- Temporising procedure only.
- Up to 25% complications including death from aspiration, migration and or perforation, 50% rebleed. Need airway protection by endotracheal intubation.

MALLORY-WEISS SYNDROME

- Occurs when a tear develops in the oesophageal mucosa.
- The tear most commonly occurs close to the oesophago-gastric junction.
- It is typically brought on by forceful retching and vomiting and is often associated with binge drinking, eating disorders and hyperemesis gravidarum.
- The haematemesis usually settles within 24-48 hours and endoscopy is rarely required.
- It should not be confused with **Boerhaave's syndrome**, which is spontaneous rupture of a non-diseased oesophagus, usually after vigorous vomiting. Patients with Boerhaave's syndrome present with respiratory collapse, subcutaneous emphysema and an acute abdomen. It is rare but when it does occur is most commonly seen in alcoholics.

II. LOWER GASTROINTESTINAL HAEMORRHAGE

DEFINITION

- Lower GI bleeding is defined as **bleeding distal to the ligament of Treitz**,
- **Acute lower GI bleeding** is of recent onset and may be severe, resulting in haemodynamic instability and decreasing haemoglobin levels.
 - **Massive lower GI bleeding** is defined by:
 - Passage of large volume of blood PR
 - Haemodynamic instability or shock
 - Initial decrease in haematocrit level of 6g/dL
 - Transfusion of at least 2 units of RCC
 - Bleeding that continues for 3 days
 - Significant re-bleeding within 1 week

CLINICAL ASSESSMENT

- **History**
 - Onset? Amount? Melena?
 - **Associated symptoms** such as fatigue, chest pain, palpitations, and shortness of breath.
 - **Any past medical history.**
 - **Certain symptoms** may help to distinguish between inflammatory, infective and malignant causes, such as abdominal pain, weight loss, fever, diarrhoea, or vomiting.
 - **Medication** use is also of relevance in particular any use of warfarin, heparin, NSAIDs and inhibitors of platelet aggregation, as some of these may require urgent reversal
- **Examination**
 - Assess the amount of blood loss and degree of shock, the possible source, and other medical conditions, which may play a role in the morbidity and mortality and potentially affect future investigations.
 - **Initial observations** such as dyspnoea, tachypnoea, tachycardia, and in particular postural drop in blood pressure are important and may indicate more significant blood loss and shock.
 - **The presence of abdominal tenderness** on examination may help to suggest that the source of bleeding is more likely to be secondary to an inflammatory disorder such as ischaemic colitis.
 - **A PR examination** MUST be performed in all those suspected of GI haemorrhage, not only to assess the stool colour/presence of blood, but also to look for anorectal lesions.

RISK ASSESSMENT

BLEED CLASSIFICATION SYSTEM

- Any patients with any of the criteria were deemed high risk.

Bleed criteria (High Risk)

- | |
|----------------------------------|
| • Ongoing Bleeding |
| • Hypotension (SBP <100) |
| • Abnormal clotting (PT >1.2sec) |
| • Altered mental status |
| • Severe co-morbidities |

- ANY patient with haemodynamic instability is high risk for severe bleeding.
- The tables below show the recommendations from the recently published (2008) SIGN guidelines on the management of GI haemorrhage in terms of admit/discharge decision-making.

Discharge if:	○ Under 60 yrs and...
	○ No evidence of haemodynamic disturbance and...
	○ No obvious of gross PR bleeding and...
	○ No obvious ano-rectal source of bleeding on PR/Sigmoidoscopy
Admit if:	○ Over 60 yrs old or...
	○ Haemodynamic disturbances or...
	○ Evidence of gross PR bleeding or...
	○ Taking aspirin or NSAID or...
	○ Significant co-morbidity

DIFFERENTIAL DIAGNOSIS OF LGIT BLEEDING:

1. Diverticular disease

2. Inflammatory Bowel Disease:

- Crohns Disease, Ulcerative Colitis, Ischaemic Colitis, Infective Colitis

3. Neoplasia

4. Benign Anorectal disease: Anal fissures, Haemorrhoids

5. Angiodysplasia

6. Other causes:

- Radiation injury, Meckels Diverticulum, Other small bowel pathology, Solitary Rectal Ulcers,
- Portal colopathy, Prostate biopsy sites, Dieulafoy lesions, Endometriosis, Colonic varices
- *10-15% of patients with an apparent lower GI bleed will in fact have an upper GI source for their bleeding*
- *Bleeding Oesophageal Varices or Peptic Ulcer Disease can present with fresh blood PR*

INVESTIGATION STRATEGIES

- **ABGs**
- **Bloods:** FBC, U&E, LFTs, Coagulation Screen, Type specific or Cross match
- **ECG**
- **PR and FOB**
- **Imaging:** CXR, PFA (to rule out other pathologies)
- **NGT:** lavage if UGIT Bleed suspected

ED MANAGEMENT OF LGIT BLEEDS

• High risk bleeds:

- Same as UGIT Bleeds (see above) & Refer to Surgery

• Low risk bleeds

- **Anal fissures** can normally be discharged home from the ED with adequate analgesia and advice regarding diet/water intake, or faecal softening agents.
- **Haemorrhoids** if otherwise stable can usually be safely discharged home from the ED with faecal softening agents. Haemorrhoids are another cause of PR bleeding. Patients with haemorrhoids typically present because of one of three complications of piles: **haemorrhage, prolapse or thrombosis**. However, they may need further investigation as an outpatient to rule out a more sinister cause for bleeding.

CHAPTER 20. HEADACHE

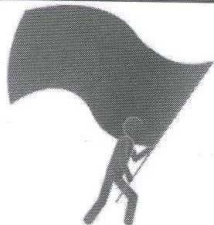
I. GENERAL APPROACH

- Headache is classified into **primary** and **secondary** groupings.
 - **Primary headaches** are those where the specific aetiology is not fully understood e.g. migraine.
 - **Secondary headaches** have a clear and understandable origin e.g. ruptured aneurysm.

CLINICAL ASSESSMENT AND RISK STRATIFICATION

- 90% of headache presentations to the ED are due to primary headaches, usually **tension headaches or migraine**. Most patients will be discharged, and many require no investigation beyond a focussed clinical history and examination.
- **Clinical history**
 - The clinical history is the single most important assessment tool when determining the cause of a headache. The most significant findings are:
 - Sudden onset – ‘**thunderclap**’ headache
 - ‘**Worst headache**’ ever
 - **New headache** in the elderly
 - **Loss of consciousness**
 - **Headache associated with activity**
 - **If any of these are present, further investigation is required.**
- **Examination**
 - The most important features of the clinical examination are:
 - Cognitive state, Vital signs
 - Neck movement, Pupils – symmetry and fundi
 - Motor function – Pronator drift
 - Gait
 - **If any of these are abnormal, further investigation is required.**
- Approximately 10% of patients will have signs or symptoms of headache due to a secondary cause.

RED FLAG SYMPTOMS



- **These are:**
 - Headache in someone >50 years
 - ‘Thunderclap’ headache
 - Headaches increasing in severity and frequency
 - Headache with fever, neck stiffness, or reduced level of consciousness
 - Focal neurological symptoms or signs
 - Papilloedema
 - Headache after trauma

- *The presence of any ‘red flag’ feature mandates further investigation of a patient presenting with headache.*

INVESTIGATIONS

- The most important investigation is the **neurological examination itself**.
- *Most patients with a normal neurological examination and a ‘non-thunderclap’ headache will require no further investigation.*

- In about 10% of ED headache patients, the history and/or the examination will suggest the possibility of a secondary cause. Such patients will need to undergo a **Brain Computerised Tomography (CT) scan**.
- CT scanning is indicated on first presentation to exclude subarachnoid haemorrhage (SAH)/structural lesion. A **CT scan** is **not** indicated in patients with symptoms of a tension-type headache, cluster headache and trigeminal neuralgia.

II. HEADACHE TYPES

HEADACHE TYPES

Primary headaches

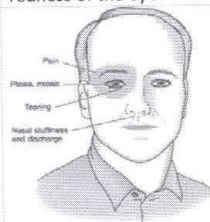
- Migraine.
- Tension-type headache.
- Cluster headache.
- Miscellaneous: Benign Cough Headache, Benign Exertional Headache, Headache associated with Sexual Activity.

Secondary headaches

- Head injury (including post-traumatic headache).
- Vascular disorders (e.g. subarachnoid haemorrhage (SAH), stroke, intracranial haematoma, cavernous sinus thrombosis, hypertension, unruptured arteriovenous malformation, temporal arteritis).
- Non-vascular disorders (e.g. idiopathic intracranial hypertension, intracranial tumour, post-lumbar puncture).
- Headaches associated with substances or their withdrawal (including analgesia, caffeine, nitrates, alcohol, and carbon monoxide).
- Infections (e.g. Encephalitis, Meningitis, Sinusitis).
- Metabolic (e.g. Hypoxia, Hypercapnea, Hypoglycaemia).
- Craniofacial disorders (e.g. pathology of skull, neck, eyes, nose, ears, sinuses, mouth, and temporomandibular joints causing pain; this includes headache secondary to glaucoma).
- Headache attributed to psychiatric disorders.
- Cranial neuralgias (e.g. trigeminal neuralgia).

CLUSTER HEADACHES

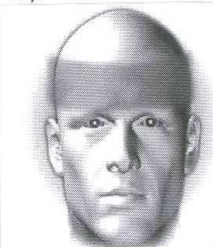
Unilateral;
Pain is in and around one Eye
Severe temporal headache
Ipsilateral rhinorrhea
Ipsilateral eye tearing
redness of the eye



CLUSTER HEADACHE

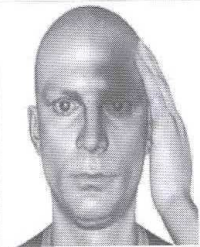
TENSION HEADACHES

Bilateral Pain is like hand/Band squeezing the head
Associated with stress in life
Occurs 3-4 times a week, mostly at the end of the day



MIGRAINE HEADACHES

Pain is **POUNDING**: Pulsatile, Onset 4-72hrs, Unilateral, Nausea & Vomiting, Disabling
Associated with aura
Lasts 2-3 hours



SINUS HEADACHES

Pain behind the forehead and/or cheekbones
Fever, Headache and nasal discharge



TYPES	CLINICAL FEATURES	MANAGEMENT
PRIMARY HEADACHES		
Migraine	<p>The best predictors for migraine can be summarised as follows:</p> <ul style="list-style-type: none"> ○ POUNDING: Pulsating, Duration of 4-72 hOurs, Unilateral, Nausea, Disabling ○ Builds up over minutes to hours. ○ Variable duration but may last up to 72h. ○ May be preceded by an aura (15-33% of patients). ○ Moderate to severe in intensity. Often disabling. ○ Associated with nausea and vomiting. ○ Exacerbated by light (photophobia), sound (phonophobia), and physical activity. Episodic (patient may have a history of previous migraines). ○ Sensitivity to light between attacks. ○ Positive family history of migraine. 	<ul style="list-style-type: none"> ○ Analgesics ○ Anti-emetics: Metoclopramide and Domperidone ○ Non-specific Therapies <ul style="list-style-type: none"> ▪ Chlorpromazine 25-50 mg IM ▪ Prochlorperazine 10mg IV/IM ○ Specific Therapies: <ul style="list-style-type: none"> ▪ The triptans: Sumatriptan 6 mg sub-cut ▪ Ergotamine tartrate 1-2 mg if migraine does not respond to triptans. <p>Prevention: Biofeedback; Propranolol, Timolol Divalproex sodium Calcium Blockers and NSAIDs</p>
Tension-type headache	<ul style="list-style-type: none"> ○ Pain is typically bilateral. ○ Pressing or tightening ('band-like') in quality. ○ Non-throbbing pain ○ Mild to moderate intensity. ○ No nausea or vomiting. ○ Not aggravated by physical activity. ○ May have pericranial tenderness. ○ May have sensitivity to light or noise. 	<ul style="list-style-type: none"> ○ Rest; Aspirin; Acetaminophen; Ibuprofen; Naproxen sodium; ○ Combinations of analgesics with caffeine; ○ Ice packs; Muscle relaxants; ○ Antidepressants, ○ Prevention: Avoidance of stress; use of biofeedback; relaxation techniques; or antidepressant medication
Cluster headache	<ul style="list-style-type: none"> ○ Severe unilateral headache. ○ Excruciating pain in the vicinity of the eye; tearing of the eye; nose congestion; and flushing of the face. ○ Pain frequently develops during sleep and may last for several hours. ○ Attacks occur every day for weeks, or even months, and then disappear for up to a year. ○ 80% of cluster patients are male, most between the ages of 20 and 50. ○ Precipitating Factors: Alcoholic beverages; excessive smoking 	<ul style="list-style-type: none"> ○ High flow O₂ therapy: 10 L/minute for 15 minutes is usually effective. ○ Sumatriptan, 6 mg, sub-cut ○ Ergotamine ○ Intranasal application of local anesthetic agent ○ Prevention: Use of steroids; ergotamine; calcium channel blockers; and lithium
Exertional Headaches	<ul style="list-style-type: none"> ○ Explosive headache indistinguishable from a SAH. ○ Related to sexual activity usually at or near orgasm. 	<ul style="list-style-type: none"> ○ Treated with Aspirin, Indomethacin, Propranolol.
Headache associated with sexual activity (coital cephalgia)	<ul style="list-style-type: none"> ○ Classically the headache is severe and throbbing. ○ The first-time a patient experiences coital cephalgia a subarachnoid haemorrhage should be actively excluded. 	<ul style="list-style-type: none"> ○ Extensive testing is necessary to determine the cause. ○ Surgery is occasionally indicated to correct the organic disease. ○ Prevention: Alternative forms of exercise; avoid jarring exercises

SECONDARY HEADACHES

Subarachnoid haemorrhage (SAH)	<ul style="list-style-type: none"> ○ Sudden-onset, 'worst-ever' headache. ○ Maximum intensity usually reached in less than 1 min. ○ Usually occipital and may be described like a blow to the back of the head. ○ May be associated with vomiting, neck pain, and photophobia. ○ The patient may present with a transient loss of consciousness or fits. ○ The patient may be drowsy and/or confused. ○ May have a history of a 'warning headache' days to weeks earlier. ○ Fundoscopy may show subhyaloid retinal haemorrhage (haemorrhage near the optic nerve head). ○ May have focal neurological deficits depending on the location of the aneurysm (e.g. IIIrd nerve palsy with posterior communicating artery aneurysms).
Meningitis	<ul style="list-style-type: none"> ○ Generalized headache in an unwell/drowsy patient. ○ May have neck stiffness and photophobia. ○ May be pyrexial. ○ May have a rash (meningococcal).
Space-occupying lesion (raised ICP)	<ul style="list-style-type: none"> ○ Headache exacerbated by lying down and valsava manoeuvres (e.g. coughing, straining, laughing, bending forwards). ○ Headache may wake the patient from sleep. ○ Visual obscurations (transient changes in vision) with change in posture or valsava suggest raised intracranial pressure. ○ Seizures, Cognitive change or focal neurological signs and Papilloedema.
Temporal arteritis	<ul style="list-style-type: none"> ○ Diffuse, throbbing headache. Patient age >50 years. ○ Scalp tenderness, jaw claudication, and tender temporal artery with reduced pulsation. ○ Visual disturbance. ○ A normal ESR makes the diagnosis unlikely. <p>Management:</p> <ul style="list-style-type: none"> ● <i>Carbamazepine, Phenytoin, Valproate, Lamotrigine and Gabapentin</i> ○ Approximately 30% of patients do not respond to drug therapy, and these patients may need surgical intervention.
Acute angle closure glaucoma	<ul style="list-style-type: none"> ○ Unilateral headache. ○ Eye pain. ○ Mid-dilated, red eye. ○ Halos around lights. ○ Reduced visual acuity.
CO2 Poisoning	<ul style="list-style-type: none"> ○ Headache that improves on leaving the environment. ○ Nausea and vomiting. ○ Dizziness, Muscle weakness and Blurred vision.

● **COMMON CAUSES OF THUNDERCLAP HEADACHES**

- SAH
- *Cerebral Venous Thrombosis*
- *Cervical Arterial Dissection*
- *Benign Exertional Headache*
- *Pituitary Apoplexia*
- *Ischaemic Stroke*
- *Hypertensive Crisis*
- *Spontaneous Intracranial Hypotension*
- *Benign Orgasmic Headache*

III. NON-TRAUMATIC SAH

AETIOLOGY

- There are three main classes of non-traumatic SAH. These are:
 - **Aneurismal SAH:** 85% (Ruptured aneurysms)
 - **Non-aneurismal SAH:** 15%
 - 10% Perimesencephalic Haemorrhages
 - 5% other: AV malformations, inflammatory, Cocaine.

PATHOPHYSIOLOGY

1. ANEURYSMS

- **NOT** Congenital
- Occur in about 1 in 40 people (1-5%).
- Most never rupture.
- Arise at sites of arterial branching.
- Multiple aneurysms in 30%.
- 90% = **Saccular** = '**Berry**' aneurysms
- 10% = **Fusiform** – usually in vertebrobasilar system and present with Cranial nerve or brainstem symptoms due to pressure effects rather than haemorrhage
- **RISK FACTORS**
 - Modifiable risk factors include:
 - *Alcohol*
 - *Smoking*
 - *Hypertension*
- These are much more important in the causation of SAH than any genetic predisposition which is implicated in only 10% of cases.

2. PERIMESENCEPHALIC HAEMORRHAGE

- **Definition** = haemorrhage restricted to the cisterns about the brainstem and suprasellar cistern **and** a negative cerebral angiogram.
- Has a much better prognosis than standard SAH with a much lower rate of rebleeding or vasospasm.
- 1 out of 29 patients rebled and died in one retrospective study.
- Has a presumed venous aetiology but some neurosurgeons are sceptical of this as an entity and advocate a repeat of the angiogram.

CLINICAL FEATURES

- **Headache:**
 - Classically presents with what's known as a '**thunderclap**' headache.
 - In reality only about 1 in 10 people who present to an Emergency department with a thunderclap headache will have had a SAH.
 - The onset may not always be instantaneous.
 - As to how short in duration a headache can be and still be a SAH, no-one knows, however an arbitrary time of **1 hour** has been suggested. The typical duration is of the order of **1-2 weeks**.

- o A '**sentinel bleed**' or **Herald bleed** is essentially a subarachnoid haemorrhage that the patient did not seek medical attention for, or one missed by a doctor. As a concept it has little or no use in the decision-making process in the emergency department.
- **OTHER FEATURES:**
 - o Vomiting is **not** predictive.
 - o Seizure at onset is.
 - o 2/3rds have a reduced level of consciousness.
 - o Neck stiffness may develop – but usually only after several hours and is due to an inflammatory reaction to the blood in the subarachnoid space, and it may not develop at all if there's only a small amount of blood.
 - o *3rd nerve palsy due to an aneurysm in the posterior communicating artery.*
 - o 1 in 7 will have intraocular haemorrhages.
 - o **Ischaemic changes (of any type) on ECG** are common
 - Possibly due to a catecholamine surge or a change in autonomic vascular tone.
 - o 3% will have a cardiac arrest
 - Aggressive resuscitation is essential as they appear to have a high rate of ROSC and half of the survivors will regain independent living.

INVESTIGATIONS

- The **most widely accepted approach** to the investigation of thunderclap headaches is a combination of CT, followed by a lumbar puncture (LP) 12 hours after onset of headache if the CT is negative.

1. CT Scan

- o Modality of choice.
- o The distribution of blood on the initial CT Head scan can be helpful in distinguishing aneurismal SAH from perimesencephalic haemorrhage.



Figure 1.20. Aneurismal SAH and Perimesencephalic haemorrhage.

- Ruptured aneurysms are most often found in the **Anterior Communicating Artery** (blood in interhemispheric fissure) (right image) followed by the **Internal Carotid Artery**, **Middle Cerebral Artery** (blood in the Sylvian fissure) and **Vertebrobasilar Circulation**.
- However, non-contrast CT brain appearances are not unique and **CT angiography (CTA)** is required in these patients to exclude a ruptured vertebrobasilar aneurysm.

2. CT ANGIOGRAPHY AND ANGIOGRAPHY

- All patients with CT-proven SAH should undergo **CTA or formal Angiography** to identify the aneurysm responsible or confirm the absence of such in cases of perimesencephalic haemorrhage. *A negative CT alone is not yet enough evidence to exclude SAH.*

3. LUMBAR PUNCTURE

- Since CT does not have 100% sensitivity, the concern is that a SAH may be missed despite a normal scan.
- Traditional teaching and expert opinion still mandate a lumbar puncture (LP) and cerebrospinal fluid (CSF) analysis for **xanthochromia** in every patient with a negative or non-diagnostic CT head scan as evidenced by national guidelines in the United Kingdom (UK) and the United States (US).
- Patients in whom the diagnosis of SAH is considered but in whom the CT is normal must subsequently undergo an **LP at least 12 hrs after the onset of symptoms.**

4. MRI SCAN

- Appears comparable to CT in acute phase
- Small studies hint it may even be better.
- May help localise 'CT negative, LP positive' patients
- May pick up pathologies not detected by CT
 - Eg: Cerebral venous sinus thrombosis (CVT), Parenchymal lesions.

ED MANAGEMENT OF SAH

• SUPPORTIVE

- **A and B:**
 - Ensure adequate oxygenation (aim for oxygen saturations >94%).
 - Aim for PaCO₂ in normal range.
 - Intubate and ventilate as required to achieve these aims and protect the airway.
 - Tape the endotracheal tube in place rather than tie it to avoid increases in ICP.
 - Avoid excessive intra-thoracic pressures to prevent rises in ICP.
- **C:**
 - Maintain end organ perfusion (aim for MAP ≥ 80 mmHg).
 - Use urine output as indicator of adequate renal perfusion.
- **D:**
 - Maintain normoglycaemia.
 - Treat seizures (benzodiazepines, prophylactic phenytoin).
 - Position—30° head-up tilt to help reduce ICP.
 - Pain management to avoid increases in ICP (if the patient has severe pain titrate morphine IV in 1-mg increments).
 - Prophylactic anti-emetics: Vomiting potentially catastrophic.
- **E:**
 - Temperature control (aim for normothermia).
 - Quiet environment with minimal visitors.
 - DVT prophylaxis
 - Stockings and pneumatic leg compression prior to intervention
 - Heparin after.
- Prophylactic laxatives and good oral fluid intake if swallow OK ('Straining at stool' is probably not a good idea!)

- **SPECIFIC**

- o May require haematoma evacuation.
- o Prevent Rebleeding
- o Prevent delayed cerebral ischaemia
 - **Nimodipine 60mg 4hourly x 3 weeks** PO or via NG (IV route unproven).
- o Seizure prophylaxis (advocated by some)

- **COMPLICATIONS OF SAH**

- o Rebleeding
- o Hydrocephalus
- o Cerebral vasospasm
- o SIADH, resulting in hyponatraemia.
- o Neurological deficits from cerebral ischaemia
- o Neurogenic pulmonary oedema
- o Aspiration pneumonia
- o Myocardial ischaemia or infarction due to excessive catecholamine release
- o Left ventricular dysfunction due to excessive catecholamine release
- o Death.

PROGNOSIS

- There are 2 scales in widespread use, despite a lack of validation:

HUNT – HESS SCALE		Survival
1	Asymptomatic / mild headache	70%
2	Moderate / severe headache; neck stiffness +/- cranial nerve palsy	60%
3	Altered mental status +/- mild focal neurological deficits	50%
4	Reduced GCS +/- hemiplegia	20%
5	Coma or decerebrate posturing	10%

WORLD FEDERATION OF NEUROLOGICAL SURGEONS SCALE

Grade 1	GCS 15
Grade 2	GCS of 13-14
Grade 3	GCS of 13-14 + motor deficit present
Grade 4	GCS of 7-12
Grade 5	GCS of 3-6

INVESTIGATIONS SUMMARY

- At present, evidence and opinion dictate that all patients presenting with acute severe headache <2 weeks from the index episode should undergo non-contrast CT scanning of the brain.
- If this is reported as normal (ideally by a neuroradiologist) then an LP should be undertaken **at least 12 hours from the start of the headache**.
- The last CSF sample should be protected from the light and transported quickly to the laboratory for analysis (by spectrophotometry in the UK) for **xanthochromia**.
- If both CT and LP are negative within two weeks, then SAH can be excluded.
- Patients presenting >2 weeks from the index headache or in whom results of either CT or LP have been unobtainable or dubious should be discussed with a neurosciences centre.

CHAPTER 21. JAUNDICE

PATHOPHYSIOLOGY

- **Pre-hepatic causes of jaundice include**
 - Anything that causes **increased rate of red cell breakdown (haemolysis)** will lead to jaundice due to **increased haem metabolism** and saturation of enzymes.
 - Malaria
 - Sick cell anaemia
 - Spherocytosis
 - Glucose 6-PD deficiency
- **Hepatic**
 - Anything that impacts on the hepatic **metabolism of bilirubin** can cause jaundice
 - **Unconjugated bilirubin** travels to the liver bound to serum albumin where it is conjugated with glucuronic acid to form conjugated bilirubin.
 - **Drugs and toxins:** Alcohol, paracetamol, anabolic steroids, isoniazid, amanita toxin, chlorpromazine, flucloxacillin, halothane.
 - **Infections:** viral hepatitis, infectious mononucleosis, leptospirosis
 - **Metabolic:** Wilsons disease, Reyes disease, haemochromatosis
 - **Granulomatous:** Wegeners granulomatosis, lymphoma, sarcoidosis, mycobacterial
 - **Genetic:** Gilberts syndrome, Crigler Najjar syndrome, Dubin-Johnson syndrome
 - **Others:** fatty liver of pregnancy, primary biliary cirrhosis, amyloidosis, metastatic carcinoma, neonatal jaundice
- **Post-Hepatic**
 - Obstructive causes are due to **inability to excrete bile**
 - **Conjugated bilirubin** is excreted into biliary and cystic ducts as part of bile.
 - In the small intestine, it is converted by enzymes to **urobilinogen**.
 - Urobilinogen can be further converted to **stercobilinogen** and passes out with faeces or reabsorbed by intestinal cells and transported in blood to the kidneys where it is oxidised to urobilin and passed out with urine.
 - **Stercobilin and urobilin** are responsible for colouration of faeces and urine respectively.
 - **Post-hepatic causes of jaundice include**
 - Drugs (amitriptylline, prochlorperazine, verapamil, co-amoxiclav)
 - Gallstones (cholecystitis alone does not produce jaundice)
 - Pancreatic carcinoma
 - Primary sclerosing cholangitis
 - Biliary atresia
 - Bile duct strictures
 - Cholangiocarcinoma
 - Pancreatitis
 - Pancreatic pseudocyst

CLINICAL ASSESSMENT

- Clinical assessment should concentrate on taking an accurate history and examination looking for findings that will help differentiate the causes of jaundice.
- The history should focus on questioning about:

- **Colour of urine and stool,**
- **Weight loss**
- **Family history of jaundice**
- **Risk factors:**
 - Alcohol intake
 - Transfusion of blood products
 - Sexual contact with a person known to have hepatitis or promiscuous sexual activity
 - Intravenous drug misuse
 - Recent tattoos or body piercing
 - Recent foreign travel
 - Accidental needle stick injury
- **A full examination**
 - Focus on the GI system as a whole may provide clues as to the cause of jaundice.
 - A hard-nodular liver on a background of known malignancy may indicate metastatic disease.
 - Signs such as *palmar erythema, spider naevi, proximal muscle wasting/weakness, hepatic flap, fetor hepaticus, cerebellar signs or encephalopathy* may indicate alcoholic liver disease.
 - **Fever and right upper quadrant tenderness in association with jaundice** are known as **Charcots Triad**, characteristic of **acute cholangitis**.
 - Painless jaundice and cachexia and an epigastric mass suggests biliary obstruction due to malignancy.

INVESTIGATION STRATEGIES

- Jaundice will be apparent when the serum bilirubin is **3x above normal**.

a. Urinalysis

- **Pre-hepatic hyperbilirubinaemia:** Unconjugated bilirubin is bound to Albumin and is not water-soluble therefore cannot appear in the urine.
- **Post-hepatic hyperbilirubinaemia:** Conjugated bilirubin is water-soluble and therefore appears in the urine.
- **Urobilinogen** is absent due the inability of conjugated bilirubin to be excreted in to the small intestine.
- The findings in the urine should then be confirmed by measuring direct (conjugated) and total bilirubin levels

Urine is NEGATIVE for Bilirubin	Urine is POSITIVE for bilirubin
↓	↓
Total Bilirubin RAISED	Direct bilirubin is RAISED
↓	↓
Direct bilirubin is NOT RAISED	↓
↓	
Investigate for: Unconjugated hyperbilirubinaemia:	Investigate for conjugated hyperbilirubinaemia
<ul style="list-style-type: none"> ● Haemolysis, ● Drug toxicity, ● Genetic disorder 	

b. Liver Function Tests

	NORMAL RANGE	NOTES
Alkaline Phosphatase	ALP: 25-115 U/L	Considerably raised in extrahepatic and intrahepatic biliary disease.
Transaminases	AST: 10-40 U/L ALT: 5-40 U/L	Usually highly raised in hepatocellular disease
Gamma Glutamyl Transferase	Male < 50 U/L Female < 32 U/L	Sensitive but not specific for alcohol intake, Raised GGT and ALP suggest cholestasis.

- **Other blood tests include:**
 - FBC, U&E, Amylase
 - Hepatitis serology
 - Autoimmune markers
 - Alpha 1 antitrypsin
 - Ferritin/transferrin saturation – Ferritin levels >1000ng/ml and transferrin saturations > 50% will indicate Haemochromatosis.
- **Imaging**
- The majority of diagnostic imaging in patients with jaundice will be performed out with the ED if the patient is stable.
 - Abdominal USS
 - CT
 - ERCP/ MRCP
 - Liver Biopsy

MANAGEMENT OF JAUNDICE IN ED

- **GENERAL MANAGEMENT OF JAUNDICE**
 - It is important to remember that jaundice can reflect a medical emergency. These cases include:
 - *Ascending cholangitis*
 - *Fulminant hepatic failure*
 - *Massive haemolysis*
 - *Neonatal jaundice*
 - ↑GGT+ ↑ALP = Cholestasis >>> Admit
 - Jaundice+ (Anaemia) Haemolysis= Admit
 - Jaundice + ↑AST/ALT= Hepatocellular injury >>> Admit if:
 - Coagulopathy
 - Sepsis
 - Altered Mental Status,
 - Intractable pain/vomiting
- **SPECIFIC MANAGEMENT OF JAUNDICE**
 - 1. Acute Liver Failure**
 - Patients with **hepatocellular injury**, **coagulopathy** and **altered mental status** may have acute liver failure and require **admission to critical care area**.
 - The most common cause of fulminant hepatic failure (FHF) in UK is **paracetamol poisoning** but **acute Hepatitis B** is the most common cause worldwide due to its prevalence.

- Prior to liver transplantation, the mortality of FHF was greater than 80%.
- The most important aspect of managing FHF is good supportive care remembering that encephalopathy may lead to failure to protect and maintain an airway.
- Fluid resuscitation and haemodynamic monitoring are also important

2. Paracetamol

- The management of paracetamol poisoning depends upon when the patient presents to the ED and the estimation of quantity of paracetamol taken
- **Within 1 hour of overdose:**
 - Give **activated charcoal** as decontamination, absorption from the gut is usually complete within 2 hours.
 - If the quantity is potentially life threatening, **gastric lavage** is thought to be useful if it can be carried out within 1 hour of ingestion.
 - Contraindications to lavage are patients who have a compromised, unprotected airway.
- **Within 8 hours of overdose:**
 - If the patient presents within 8 hours, **N-Acetyl Cysteine** is proven to reduce the risk of serious hepatotoxicity, however when the patient presents as late as 48 hours, NAC is still beneficial.
 - **Indications for consideration of liver transplant are:**
 - Acidaemia (pH <7.3)
 - Renal insufficiency
 - Grade III or worse hepatic encephalopathy
 - Elevated PT

3. Pregnancy and neonatal jaundice

- Involve obstetrician/ Paediatrician.
- Patients who present with jaundice in the third trimester may require **delivery**.
- Well appearing neonates with a bilirubin <15mg/dL can safely be discharged home with close outpatient follow-up.
- Neonatal jaundice can often be physiological due to increased break down of premature erythrocytes and insufficient Glucuronyl Transferase in the newborn liver but jaundice persisting after 2 weeks requires investigation.
- Neonatal jaundice is treated with **phototherapy**.
- **Exchange transfusion** is an aggressive treatment to lower bilirubin levels.

Key Learning Points

- Understanding Haem metabolism is key to understanding jaundice.
- The differential diagnosis of jaundice is very wide.
- **LFTs** are the key Emergency department investigation in jaundice.
- **Abdominal USS** is useful and readily available as first line imaging in the ED.
- Patients with coagulopathy, sepsis or altered mental status must be admitted.
- Ascending cholangitis, fulminant hepatic failure, massive haemolysis and neonatal jaundice reflect medical emergencies

CHAPTER 22. ATRAUMATIC LIMB PAIN

I. CARPAL TUNNEL SYNDROME

- Median nerve compression at the wrist caused by the transverse carpal ligament is common and usually occurs in women.
- **HISTORY**
 - Carpal tunnel syndrome is most often unassociated with trauma, and patients report the gradual onset of primarily nocturnal hand, wrist, and forearm pain often accompanied by numbness or dysesthesias.
 - Pain is sometimes better localized to the volar first or second fingers. Patients are commonly awakened from sleep and report relief of symptoms by shaking or elevating the hands. Bilateral involvement is occasionally reported, but more often only one upper extremity is involved.
 - The incidence is increased in pregnancy and associated with birth control pill use.
 - The diagnostic impression of carpal tunnel syndrome can be further supported if holding the patient's wrist in flexion (**Phalen test**) for 60 seconds reproduces symptoms and placing the wrist in the neutral position relieves symptoms.
 - **Tinel sign** (light tapping over the median nerve as it crosses under the carpal ligament) may also elicit symptoms (tingling in the fingers in the median nerve distribution) and is useful diagnostically.
- **DIAGNOSTIC TESTS**
 - Radiologic assessment should be undertaken when trauma has preceded the onset of symptoms, because **carpal displacement** and **Colles fractures** have both been associated with the development of the carpal tunnel syndrome.
 - More commonly, tenosynovitis localized to the wrist flexors is responsible.
 - The diagnosis can be confirmed by **electromyography**.
- **TREATMENT**
 - Patients with abnormalities of motor function in the distribution of the median nerve require prompt **orthopaedic consultation** for possible decompression.
 - Other patients should be treated with **wrist immobilization by splinting in the neutral position**; patients should remove the splint once each day for bathing, but it should otherwise remain applied.
 - Wearing the splint during sleep, particularly during the first 3 to 5 days of therapy, and keeping the involved extremity elevated as much as possible should be emphasized.
 - An initial trial of a **nonsteroidal anti-inflammatory agent** is recommended; treatment with **steroids** or **definitive repair** (release of the transverse carpal ligament) or both may be undertaken subsequently in selected patients.
 - Follow-up in patients without motor loss should be advised in 7 to 10 days.

II. ACUTE LIMB ISCHAEMIA

INTRODUCTION

- Acute limb ischaemia is defined as any sudden decrease in limb perfusion causing a potential threat to limb viability. By convention this usually refers to patients presenting with symptoms for less than 2 weeks.
- The spectrum of acute limb ischaemia therefore ranges from the patient with a few hours history of a painful cold white leg, to the patient with a few days history of short distance claudication or the patient with a sudden increase in ischaemic symptoms on a background of peripheral arterial disease.
- There is also some evidence that the proportion of acute limb ischaemia caused by **embolic disease** is falling, due to the decreased incidence of rheumatic heart disease and the improvement in the management of atrial fibrillation.
- Most acute limb ischaemia now occurs on a background of **peripheral arterial disease**.
- Acute limb ischaemia carries a high morbidity and mortality.

PATHOPHYSIOLOGY

- An **embolus** is defined as a material (gas, solid or liquid) that is carried within the circulation and lodges in a blood vessel in another part of the circulation, causing occlusion of the blood vessel. Radiologically the upper border of an embolus is classically concave, known as the **meniscus sign**.
- In acute limb ischaemia emboli most commonly arise from the heart (80%) and as such are usually composed of platelets.
- They can also arise from proximal arterial disease (either aneurysms or stenoses) and may then contain atheroma.
- These carry a poorer prognosis for the limb since they are harder to treat and not amenable to thrombolysis.
- **Thrombosis** may be influenced by any of the three factors described in Virchows Triad:
 - Damage to the endothelium (e.g. atherosclerosis)
 - Alteration to the blood flow (e.g. hypodynamic states such as heart failure or shock)
 - Change in the constituents of the blood (prothrombotic states e.g. underlying malignancy, haemoproliferative disorders, smoking)
- The most common cause of thrombotic limb ischaemia is **thrombosis** of a vessel on a background of **atherosclerosis**; smoking increases this risk due to its prothrombotic effect on platelets.
- **Rarer causes of acute limb ischaemia are:**
 - Iatrogenic
 - Graft occlusion
 - Aortic dissection
 - Vasculitis
 - Popliteal entrapment syndrome
 - Compartment syndrome

CLINICAL ASSESSMENT

- The Classic presentation the **6Ps**:

- o **Pain:** In most cases this will occur **at rest**, although a patient with a viable limb may present with acute onset short distance claudication. **Rest pain** is usually worse in the most distal part of the limb (toes) since this has the worst perfusion, and may be relieved on dependency (hanging legs over bed). Pain which is worse on passive movement of the muscles indicates potential compartment syndrome (see below) and is a poor prognostic sign.
- o **Pallor:** this is especially useful in comparison to the opposite limb; it is also useful to check venous filling. Acutely ischaemic limbs are classically white rather than blue. Chronic critically ischaemic limbs may appear pink due to compensatory vasodilation – the so-called **sunset foot**. In this situation **Buerger's test** may also be useful (pallor on elevation of the limb, with erythema on dependency).
- o **Paraesthesia:** this is present in over 50% cases. Sensory nerves are smaller than motor nerves and more sensitive to ischaemia so tend to be affected first.
- o **Paralysis:** this is a poor prognostic sign and indicates an element of irreversible ischaemia.
- o **Perishingly cold:** this is a useful sign if used in comparison to the opposite (normal) limb. Check temperature using the back of your hand.
- o **Pulselessness** checking pulses is notoriously unreliable. Arterial Doppler signals should be checked in anyone with suspected acute limb ischaemia. ***Audible arterial Doppler signals do not eliminate the diagnosis of acute limb ischaemia.***

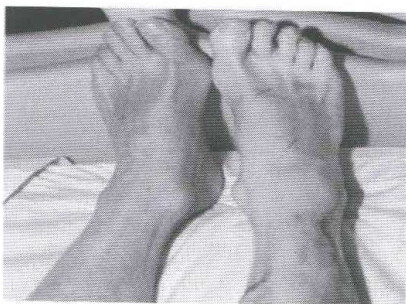


Figure 1.22.1 White suggests acute ischaemia.



Figure 1.22.2. **Fixed mottling** of the leg is a poor prognostic sign and implies irreversible ischaemia.

EXAMINATION FINDINGS

- **Cardiovascular examination**
 - o A full cardiovascular examination should be performed, in particular to detect cardiac arrhythmias or possible valve disease as a source of emboli.
 - o The abdomen should be assessed for evidence of an abdominal aortic aneurysm.

THE AFFECTED LEG

- **Inspection**
 - o **Colour**
 - **White** suggests acute ischaemia.
 - **Pink or blue** suggest Chronic ischaemia
 - **Fixed mottling** of the leg is a poor prognostic sign and implies irreversible ischaemia.

- **Dry gangrene** (black tissue) is also a late sign and consistent with chronic irreversible ischaemia (more than 2 weeks).
- **Scars** – Look for scars of previous surgery. Surgery on the abdominal aorta may be via a midline or transverse incision, patients who have had an EVAR (endovascular abdominal aneurysm repair) will only have scars on the groin. Don't forget behind the knee patients who have had a popliteal aneurysm repair may have a vertical scar behind the knee.
- **Palpation**
 - **Temperature** – Always compare to the opposite leg. It may also be helpful to assess the temperature of other peripheries (hands) and check the core temperature.
 - **Pulses** It is particularly important to determine whether the patient has a palpable **femoral pulse**.
 - **Tenderness** – Is the limb tender? This again is a poor prognostic sign as it suggests muscle ischaemia. Is there pain on passive movement? This suggests compartment syndrome and requires immediate vascular referral for urgent intervention.
 - **Neurological function** – Test sensory and motor function. Loss of sensation is common. Loss of motor function is a poor prognostic sign. Any neurological deficit implies the need for emergency intervention.
- **Auscultation**
 - Arterial Doppler signals
- **The Other Leg**
 - It is essential to fully examine both legs, the comparison between the normal and abnormal leg will often aid both diagnosis and determining probable aetiology.

RUTHERFORD'S CLASSIFICATION OF ACUTE LIMB ISCHAEMIA

	Capillary return	Motor	Sensory	Arterial Doppler signal	Venous Doppler signal
I: Viable					
Ila: Threatened (salvageable if promptly treated)	Intact/slow		Partial (toes only) or none	x (often)	
Ilb: Threatened (salvageable with immediate reconstruction)	Slow/absent	Partial paralysis	Partial (more than toes) or complete	x (usually)	
III: Irreversible (major tissue loss or permanent nerve damage inevitable)	Absent + staining	x profound paralysis (rigor)	x profound (anaesthetic)	x	x

DIFFERENTIAL DIAGNOSIS

- Compartment Syndrome
- Cerebrovascular accident (CVA)
- Deep vein thrombosis (DVT)
- Hypovolaemic Shock
- Acute compressive neuropathy

INVESTIGATION STRATEGIES

- **ECG:** To diagnose AFib or other cardiac arrhythmias which may be a source of emboli.
- **Bloods:**
 - o Full blood count
 - o Urea and Electrolytes
 - o Glucose
 - o Creatinine Kinase
 - o Clotting
 - o Group and Save
- **Arterial Blood Gas analysis.**
- **Imaging:** The urgency of imaging depends on the presentation.
 - o A Digital Subtraction **Angiogram**.
 - o **MR angiography** and **CT angiography** are less invasive and should provide the same anatomical information.
 - o **Arterial duplex** is non-invasive but is operator dependent and iliac and calf vessels can be difficult to image.
 - o The choice of imaging is likely to depend on the local resources available.

ED MANAGEMENT OF ACUTE LIMB ISCHAEMIA

- **Initial management in the Emergency Department**
 - o **Analgesia:** *IV morphine*. As with any painful condition there is no rationale to withhold analgesia in order to facilitate assessment.
 - o **Oxygen:** all patients should be administered supplemental oxygen.
 - o **HEPARIN:** **5000units intravenous heparin** (unfractionated) should be given immediately to all patients with acute limb ischaemia; even they are likely to be undergoing surgery or angiography. This is to prevent propagation of thrombosis. In patients in whom definitive treatment is deferred an intravenous heparin infusion should be prescribed.
 - o **IV FLUIDS:** Patients with acute limb ischaemia are often dehydrated. In addition, they are likely to be undergoing surgery or being given iodinated contrast which will be a further renal insult. Reperfusion of ischaemic tissue releases toxic metabolites, potassium, creatinine kinase and myoglobin which can further damage the kidneys. **Administration of potassium should be avoided.**
 - o **REFER:** Refer to a vascular specialist urgently. Any delay risks jeopardising the limb, particularly if there is sensorimotor impairment

II. ACUTE CRYSTAL ARTHROPATHY (GOUT & PSEUDOGOUT)

- This will account for a large proportion of the cases of true monoarthropathy seen in the ED. The age range is usually 40–70 years and risk factors include excess alcohol consumption, male sex, obesity, renal impairment and drugs (low-dose aspirin and diuretics). **A raised serum urate level** is neither specific nor sensitive enough to assist in the diagnosis, since it is often normal in acute episodes of gout.
- Other blood tests (FBC and CRP) that are often performed in the ED in attempts to differentiate the causes of acute monoarthropathy are equally lacking in sensitivity and specificity.

- The **diagnosis** of crystal arthropathy is confirmed by the presence of birefringent crystals viewed on polarized light microscopy.
- **Treatment options** in the acute phase include intra-articular steroid injection, non-steroidal anti-inflammatory drugs (NSAIDs) or colchicine.

III. REACTIVE ARTHRITIS

- This usually develops 2–4 weeks after a genitourinary (*Chlamydia*) or gastrointestinal (*Shigella*, *Salmonella* or *Campylobacter*) infection, although approximately 10% of cases do not have a preceding symptomatic infection.
- Only a minority of patients will have the classical triad (**conjunctivitis, urethritis and arthritis**) described by **Reiter**.
- The age range is typically 2–40 years. Onset is usually acute and can resemble septic arthritis, but will often affect more than one joint.
- Elevated white cell count (WCC) and CRP are common, and joint aspiration will be needed to exclude septic arthritis.
- The disease is self-limiting in 3–12 months, but will require management with NSAIDs or steroids (either systemic or intra-articular).

IV. SEPTIC ARTHRITIS

- This is relatively rare, but must always be considered, since missed diagnosis has the potential to cause devastating joint destruction (up to 50%).
- **Risk factors include:**
 - *Chronic arthritides (especially rheumatoid arthritis),*
 - *Previous surgery (especially prosthetic joints),*
 - *Immunosuppression,*
 - *Extremes of age,*
 - *Diabetes*
 - *Intravenous drug abuse.*
- **ETIOLOGY AND PATHOPHYSIOLOGY**
 - **Organisms may invade the joint by:**
 - Direct inoculation,
 - Contiguous spread from infected periarticular tissue,
 - Via the bloodstream (Haematogenous is the most common route).
 - Septic arthritis is caused by invasion of bacteria, viruses or fungi into the synovial membrane of a joint:
 - **Staphylococcus aureus:** is the most common cause in adults. has specific affinity of synovial structures.
 - **Streptococci:** the second most cause
 - **Haemophilus influenza:** was the most common in children but is now uncommon in areas where Hib vaccination is practiced.
 - **Neisseria gonorrhoea:** in young adults, multiples macules or vesicles seen over the trunk are pathognomonic features.
 - **Escherichia coli:** in elderly, IV Drug users and seriously ill.
 - **M. tuberculosis:** occurs most commonly by direct inoculation, penetrating wound, or direct extension. The most common mechanism of infection is via haematogenous.

CLINICAL FEATURES OF SEPTIC ARTHRITIS

- A painful, hot, swollen, red joint is the classic presentation. Usually only one joint is affected. Only very limited movement of the joint is possible and it is usually held slightly flexed. The patient may be systemically unwell with fever and rigors. The use of analgesics, steroids, or antibiotics may obscure some of the clinical features.
- The commonest joint affected is the **knee (50%)**, followed by the **hip (20%)**, **shoulder (8%)**, **ankle (7%)**, and **wrist (7%)**.
- Detection of septic arthritis in the hip can be very difficult owing to the lack of obvious external findings due to its deep location.
- *Patients who are intravenous drug-users may have involvement of atypical joints, e.g. vertebral, sacroiliac, or sterno-clavicular joints. (Frcem Exam Question)*

INVESTIGATIONS FOR SEPTIC ARTHRITIS

- **Joint aspiration and synovial fluid analysis:** (most important diagnostic test): Fluid should be sent for **gram stain, cultures, crystal examination, and cell count**.
- **FBC, ESR, and CRP: negative results** do not rule the disease out.
- **Blood cultures:** useful in identifying the organism but do not help confirm or exclude the diagnosis in the ED.
- **X-ray:** used as useful baseline, can be initially normal.
- **Lateral X-rays** may show bone destruction.

MANAGEMENT OF SEPTIC ARTHRITIS IN THE ED

- IV Antibiotics: **Flucloxacillin and benzylpenicillin**.
- **Analgesia:** consider splintage in addition to pharmacological treatment.
- **Urgent orthopaedic referral:** for joint irrigation/drainage.

V. GONOCOCCAL ARTHRITIS

- Septic arthritis due to bacterial infections is commonly classified as either gonococcal or nongonococcal. ***Neisseria gonorrhoeae*** remains the most common pathogen (75% of cases) among **younger sexually active individuals**.
- ***Staphylococcus aureus*** infection is the cause of the vast majority of cases of acute bacterial arthritis in adults and in children older than 2 years.
- The increased incidence of this pathogen parallels the increase in presence of prosthetic joints and in the use of immunosuppressive agents.
- This pathogen is the cause in 80% of infected joints affected by rheumatoid arthritis.
- *Gonococcus* may cause two types of arthritis:
 - A **localized septic arthritis affecting one joint**.
 - An **arthritis-dermatitis syndrome** (*classic triad is dermatitis, tenosynovitis, and migratory polyarthrits*).

MANAGEMENT OF GONOCOCCAL ARTHRITIS IN THE ED

- It should be managed as any other septic arthritis, with joint irrigation and antibiotics.
- *Investigations are the same as above but should also include swabs of the urethra, cervix, throat, and rectum to help identify the causative agent.*
- Treatment is with broad-spectrum antibiotics until the causative agent is identified.
- **Cephalosporins** are appropriate once gonococcus is confirmed.
- Open drainage of affected joints is rarely required. Patients should be advised that they and **their partner(s)** require a full sexual health screen.

VI. SOFT TISSUE INFECTIONS

INTRODUCTION

- Impetigo, erysipelas, cellulitis and necrotising fasciitis represent a spectrum of soft tissue infections.

1. IMPETIGO

• AETIOLOGY

- Impetigo is a superficial infection of the **epidermis**. It is caused by **Staphylococcus aureus** and **Group A beta-haemolytic streptococci** (GABHS, also called Streptococcus pyogenes). Treatment needs to cover both organisms as they often co-exist.
- 30% of population carry Staphylococcus aureus in the anterior nares and may get recurrent impetigo around the face. Children may carry it around the perineum where it can cause a desquamating infection.
- Up to 90% of patients with atopic eczema are chronically infected with Staphylococcus aureus which may be responsible for eczema flares.
- Burns may be secondarily infected by Staphylococcus aureus, delaying healing.
- Methicillin-resistant Staphylococcus aureus (MRSA), either hospital or community acquired, is now a frequent cause of impetigo, especially with folliculitis or abscesses.

• CLINICAL FEATURES

- Several clinical forms of impetigo exist.
- All forms are more common in children, with the bullous form seen mainly in the under twos and the non-bullous two to five-year-olds.
- Adult contacts may develop crusting impetigo or folliculitis around shaved areas on the face and axillae. Systemic upset is rarely seen.
- **Complications:** Rare and seen mainly in neonates or immunosuppressed patients:
 - Meningitis
 - Sepsis
 - Secondary cellulitis
 - Pneumonia
 - Septic arthritis
 - Post-streptococcal glomerulonephritis is occasionally seen in young children.

CLINICAL FORMS OF IMPETIGO

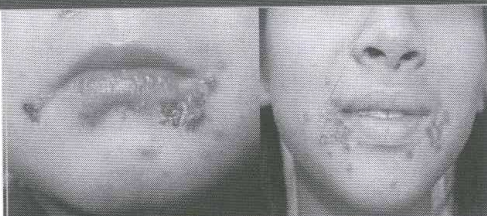
1. Non-bullous impetigo

It is the usual form.

Red macules form initially, then golden crusts.

It is itchy but not painful.

Regional lymphadenopathy is common



2. Bullous impetigo.

Here there is sloughing of the epidermis due to toxin production.

Vesicles/bullae may be on face, buttocks, nappy area or trunk.



3. Folliculitis

It is infection of the hair follicles due to *Staphylococcus aureus*.

4. Ecthyma

It is deeper, ulcerating & associated with lymphadenitis.

5. Impetiginous dermatitis

Secondary infection of pre-existing skin disease or traumatized skin



INVESTIGATION

- Impetigo is diagnosed clinically.
- However, consider **swabbing for culture and sensitivities** in the following:
 - **Suspected MRSA:** take swab from under crusts
 - **Recurrent episodes:** swab anterior nares, possibly axillae/perineum
 - **Infected eczema:** wet-swab inflamed areas

ED MANAGEMENT OF IMPETIGO

- **Use topical antibiotics** for localised areas. Clean crust before applying.
 - **Mupirocin 2%** is the best choice. Use mupirocin nasal ointment to eradicate nasal carriage when treating impetigo on the face
 - **Fucidin**, though often used, is frequently ineffective due to resistance.
- **Systemic antibiotics (generally flucloxacillin)** are needed for extensive areas and infected dermatitis
- **Inpatient care:** is required for infants with bullous impetigo and patients with widespread impetiginised dermatitis who may develop sepsis or dehydration.
- Neonates have a much higher incidence of developing sepsis and meningitis so require **paediatric referral**.
- **Follow-up** is required if lesions have not cleared in seven days
- Children should not return to daycare or school until the lesions clear
- Carers should avoid contact with lesions, towels etc. Wash clothing, toys and hands frequently

2. CELLULITIS AND ERYSIPELAS• **Aetiology**

- o Cellulitis is an acute, spreading bacterial infection of the **dermis and subcutaneous tissue**, usually complicating a wound, ulcer, or dermatitis. Affected skin becomes tender, warm, erythematous, and swollen.
- o Any age group may be affected & numerous organisms can cause it.

COMMON CAUSES OF CELLULITIS	TYPE OF INFECTION
Streptococcus pyogenes Staphylococcus aureus	<ul style="list-style-type: none"> • Cellulitis or erysipelas • Abscesses, penetrating trauma • IV-line sepsis, IV Drug abuse • Secondary infections of damaged skin (Dermatitis, burns)
Atypical organisms	Common source of organisms
Pasteurella multocida	Cat bites
Vibrio vulnificus	Sea water
Aeromonas hydrophilia	Fresh water
Streptococcusiinae	Fish farms

- **Atypical organisms** can cause severe, rapidly progressive cellulitis with marked systemic features.
- **Erysipelas** is a superficial cellulitis with invasion of the lymphatics.
 - o It is generally caused by Streptococci, especially **Streptococcus pyogenes**.
 - o Lesions are classically well-demarcated, fiery red and raised.
 - o A “**peau d’orange appearance**” (dimpling) is due to tethering of hair follicles within the oedematous dermis. Infants and the elderly are more commonly affected.



Fig 1.22.3. Cellulitis



Fig 1.22.4. Erysipelas

- Cellulitis and Erysipelas are often used interchangeably and clinical differentiation may be difficult. In both conditions, patients may be systemically well or unwell.
- **Cellulitis severity is graded I-IV:**

Class I	No systemic toxicity and no uncontrolled co-morbidity
Class II	Systemically ill or co-morbidity complicating infection
Class III	Signs of marked systemic illness (confusion, tachycardia, hypotension) or severe co-morbidity
Class IV	Sepsis syndrome or life-threatening infection such as necrotising fasciitis

Clinical features

- Both erysipelas and cellulitis present with the following:
 - **Type of lesion:** Red, oedematous tender spreading areas, which are well demarcated in erysipelas but diffuse in cellulitis.
 - There may be small haemorrhagic areas.
 - Sometimes lymphangitis and regional lymphadenopathy.
 - Vesicles/bullae are fairly common.
 - Entry wound, bite, septic source or pre-existing skin pathology such as venous eczema or athlete's foot is often apparent.
- **Systemic features**
 - Usually mild: Fever, tachycardia, confusion, hypotension, and leukocytosis are sometimes present and may precede visible skin changes.
- **Anatomic location**
 - Commonest site is the leg usually unilateral, but bilateral cellulitis does occur rarely.
 - Arm and breast cellulitis occurs after mastectomy.
- **Predisposing factors include:**
 - Diabetes
 - Immunodeficiency (more atypical organisms, eg *Pseudomonas aeruginosa*)
 - Varicella infection
 - Systemic illness
 - Impaired peripheral circulation (arterial or venous insufficiency)
 - Lymphoedema
 - Obesity

DIFFERENTIAL DIAGNOSIS OF CELLULITIS

- *Post-phlebotic limb*
- *Panniculitis*
- *Leg eczema*
- *Venous insufficiency*
- *Thrombophlebitis*
- *Deep Vein Thrombosis*

INVESTIGATIONS

- Patients with Class I cellulitis can be managed in the community with oral antibiotics.
 - **Swab** any broken skin.
 - **Culture** blister fluid.

ED MANAGEMENT OF CELLULITIS

- Admission for intravenous antibiotics is recommended for patients with:
 - Systemic upset (fever, nausea, malaise)
 - Immunosuppression (due to disease, medication)
 - Haematological malignancy
 - Co-morbidity (cardiac failure, diabetes mellitus, renal impairment)
 - Factors affecting healing (IV drug abuse, obesity, peripheral vascular disease)
 - Age extremes (below 1 year or elderly)
 - Facial and orbital cellulitis: these require urgent assessment by the appropriate specialities because of the high risk of local complications.

- **General measures**

- o The affected limb should be **elevated** and a bed cradle used
- o **Analgesia and antipyretics** as required
- o Maintain **good hydration**
- o **Mark the extent** of erythema present on admission
- o **Non-adherent saline dressings** for weeping areas

- **Choice of antibiotics**

- o **Beta-haemolytic streptococci or Staphylococcus aureus** cause almost all infections, so therapy must cover these.
 - **Flucloxacillin** is bacteriocidal against both organisms so is recommended as monotherapy for Class I (mild) infections at 500mg QDS and for moderate infections at a dose of 2gm qds.
- o Patients with Class IV infections need broad spectrum intravenous cover according to local guidelines (eg **benzylpenicillin and ciprofloxacin**).
- o **Co-amoxiclav** has a broad spectrum of activity and is therefore recommended for patients with cellulitis from bites (at a dose of 625mg TDS).
- o **Ciprofloxacin 750mg bd** should be added to flucloxacillin to cover fresh water infections.

- **Recurrent cellulitis**

- o 29% of patients admitted with cellulitis have a recurrent episode within three years.
- o Recurrence is associated with chronic lymphoedema and venous eczema.
- o Antibiotic prophylaxis should be considered for patients with recurrent cellulitis.
- o **Penicillin V 250mg bd or Erythromycin 250mg bd** have shown benefit in several small studies.

3. PANNICULITIS

- o Panniculitis refers to a group of conditions that involve inflammation of the fat under the skin, with or without associated vasculitis.
- o Despite having very diverse causes, most forms of panniculitis have the same clinical appearance.
- o Affected skin feels **thickened and woody to touch**.
- o There may be discolouration of the overlying skin, either reddening or darker, brownish pigmentation.
- o The area is often tender. Most often, the affected areas appear as raised nodules or lumps under the skin, but may be a plaque or large flat area of thickened skin.
- o Purpuric discoloration may be present and there may also be bullae and erosions.
- o Causes include the collagen vascular disorders such as **Rheumatoid Arthritis, Sarcoidosis, Polyarteritis Nodosa**, also **Crohn's Disease, Steroid Therapy, Necrobiosis Lipoidica and Erythema Nodosum**.

4. NECROTISING FASCIITIS

- **DEFINITION**

- o Necrotising fasciitis is a rapidly spreading infection of the subcutaneous tissues, with characteristic widespread fascial destruction.
- o There may also be necrosis of the underlying muscle, depending on the organisms involved.

- o It may be caused by single bacterial species (***Clostridium perfringens* or Group A Streptococci, Bacteroides and Staphylococcus aureus**), or be polymicrobial.
- o Anaerobic organisms are often present in addition to aerobes and can form gas (**gas gangrene**).
- o A saltwater variant also exists, in which a minor skin wound becomes infected with a vibrio species.
- o All varieties are rapidly progressive, resulting in massive tissue loss, multiorgan failure and death if not rapidly treated with surgical debridement.
- o Initially diagnosis may be very difficult with presentations similar to cellulitis so a high clinical index of suspicion is required.



Fig 1.22.5. Necrotising fasciitis.

AETIOLOGY AND PREDISPOSING FACTORS

- Groups at increased risk of necrotising fasciitis include **IV drug abusers, alcoholics**, those with **immunosuppression, chronic disease** and **haematological malignancies**.
- However, half of cases occur in young and previously healthy individuals.

CLINICAL FEATURES

- Early differentiation of necrotising fasciitis from cellulitis is challenging.
- However, delay in diagnosis results in higher mortality, so early clinical diagnosis is the priority.
- There are features which would point you towards a diagnosis of necrotising fasciitis:

a) Patient complains of severe pain but there is no visible skin change:

- This is one of the best early diagnostic features. Pain may be severe before any skin changes are seen, & very marked tenderness may be present, despite apparently normal skin appearance, due to the deep nature of the infection. Spread is characteristically rapid and response to antibiotics poor. Systemic toxicity develops, manifested by high fever, hypotension, leucocytosis, delirium, and renal failure.
- Later, more obvious, signs are visible skin bruising, then necrosis, and gas in the tissues detected on palpation or imaging (eg x-ray).

b) Increasing discomfort 48 hours after liposuction, necrotising fasciitis is well recognised complication of liposuction

c) Laboratory marker, the following features are associated with necrotising fasciitis:

- o *White cell count of > 14*
- o *Hyponatraemia (< 135)*
- o *Raised urea (> 15)*
- o *CRP > 16*
- o *CK > 600*
- Bullae and pre-existing diabetes are not helpful in distinguishing cellulitis from necrotising fasciitis.
- **Bullae alone** are not diagnostic of deep infections, because they also occur with erysipelas, cellulitis, scalded skin syndrome, disseminated intravascular coagulation, purpura fulminans, some toxins, and primary bullous dermatological conditions.
- While diabetics are at greater risk of necrotising fasciitis, they are also at greater risk of all soft tissue infections, especially cellulitis. Diabetics with cellulitis may also have osteomyelitis or septic arthritis especially when their cellulitis is secondary to chronic skin ulceration.

INVESTIGATION AND MANAGEMENT

- **Blood cultures** and **arterial blood gases** should be performed
- **Empiric broad spectrum antibiotics** should be administered immediately
- Other investigations that may assist diagnosis are **plain x-ray** (showing gas in tissues) and **CT** (showing gas dissecting along fascial planes)
- However, any suspicion of necrotising fasciitis requires **immediate surgical referral** for debridement and exploration in theatre
- The patient will also require **intensive care**, so early involvement of intensivists is necessary.

Key Learning Points

- Severe pain with minimal skin changes is suggestive of necrotising fasciitis.
- Blood cultures are only indicated in class III or class IV cellulitis.
- Use mupirocin nasal ointment to eradicate nasal carriage when treating impetigo on the face.
- Don't forget MRSA as a cause of infection not responding to treatment.

CHAPTER 23. NON-TRAUMATIC NECK PAIN

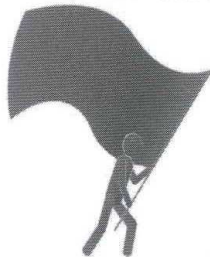
RISK STRATIFICATION

- The emergency clinician will frequently be presented with patients with neck pain without a history of trauma. Most will have simple conditions, such as spondylosis and do not pose a diagnostic challenge, nor will they need imaging.
- When neurological involvement occurs, imaging is required. High risk patients are those with **insidious onset of symptoms**, especially when other significant co-morbidities exist such as diabetes mellitus and immuno-suppression.

CLINICAL ASSESSMENT

History

- This should include timing of the onset of symptoms. Non-traumatic neck pain is always of concern, and the covert causes must be considered, including **meningitis, spinal infection and metastatic deposits within the spine.**
- **RED FLAGS IN SPONTANEOUS NECK PAIN:**



- Presentation in patients less than 20 or over 55 years of age
- Constant, progressive pain
- Past history of carcinoma
- Systemic steroids
- Drug abuse, HIV
- Systemically unwell
- Weight loss
- Persisting severe restriction of cervical flexion
- Inflammatory disorders such as ankylosing spondylitis and rheumatoid disease

Examination

- In a walking patient, follow the trusted approach: Look, Feel, Move, before imaging.

CAUSES OF NON-TRAUMATIC NECK PAIN

COMMON AND POTENTIALLY LIFE THREATENING CAUSES	UNCOMMON BUT LIFE THREATENING CAUSES
<ul style="list-style-type: none"> ◦ Acute wry neck / torticollis ◦ Spondylosis ◦ Disc impingement ◦ Spinal stenosis ◦ Meningitis 	<ul style="list-style-type: none"> ◦ Pharyngitis ◦ Infective discitis ◦ Spinal osteomyelitis ◦ Metastases

1. ACUTE WRY NECK / TORTICOLLIS

- This commonly affects adolescents and young adults.
- The patient presents with the neck held at an angle, in constant pain.
- The pain experienced tends to be localized to the mid cervical region and is unilateral away from the direction of the deformity.
- The patient often describes a history of a sudden unguarded movement of the neck which causes sudden pain and restricted neck movement.
- There is normally no history of trauma.
- **Treatment is expectant, and NSAID together with heat help to ease it.**

2. CERVICAL SPONDYLOSIS

- Cervical spondylosis refers to degenerative changes of the cervical spine; these include osteophyte formation, thickening of associated spinal ligaments, and narrowing of the intervertebral disk space.
- Although these changes are commonly noted in asymptomatic patients, they may also be associated with a variety of clinical presentations.
- This is extremely common in the over 50s, and usually causes no significant symptoms.
- Minor injury, such as a missed step, can cause an exacerbation resulting in pain, which appears disproportionate to the trivial injury.
- ***Distinction should be made between compression of the cervical spinal cord, resulting in myelopathy, and compression of spinal nerve roots, resulting in radiculopathy.***
- Both syndromes may result from bony osteophyte formation and ligamentous hypertrophy. Both may produce symptoms as a result of minor or major cervical trauma.

A. MYELOPATHY

- Myelopathy most often occurs in patients with a presumptive congenital narrowing of the cervical spinal canal.
- Symptoms related to myelopathy include *mild upper extremity weakness, atrophy, hyperreflexia in the lower extremities, and extensor plantar responses.*

B. RADICULOPATHY

- Patients with radiculopathy present with symptoms referable to the particular nerve root that is compressed; most commonly C6 and C7 are involved and result in neck, parascapular, and arm pain, all of which may be accentuated or precipitated by movement of the head or neck.
- **Motor abnormalities;** including *weakness and diminution or loss of reflexes*, may be noted in the biceps, brachioradialis, and triceps muscles.
- **Sensory loss** may involve the *radial aspect of the thumb or index and long fingers.*
- **Diagnostic Studies**
 - The demonstration of cervical spondylotic changes by plain radiography cannot be considered diagnostic, given the extremely high incidence of asymptomatic patients.
 - Such radiographic abnormalities, however, when correlated with physical findings, are suggestive.
 - The diagnosis of cervical myelopathy requires demonstration that the cervical canal is less than 10 mm in diameter; this dimension may be measured by **MRI** or **CT** imaging, which can usually be done non-emergently.
- **Treatment**
 - **Myelopathy**
 - Discussed with the **orthopaedic or neurosurgical consultant** before disposition; this is particularly true when trauma has precipitated or worsened symptoms or when motor loss is suspected.
 - **Radiculopathy**
 - **Immobilization of the neck** in a soft cervical collar
 - Several days of activity limitations;
 - NSAIDs, Local Heat, Muscle Relaxants
 - *When motor abnormalities are noted, consultation before disposition is recommended.*

3. DISC IMPINGEMENT

- In cervical disc prolapse part of the nucleus pulposus may protrude through the annulus fibrosus at its weakest part, most commonly posterolateral.
- A slight protrusion bulges against the posterior longitudinal ligament and causes localized neck pain but if larger then it may impinge upon the spinal nerve root at that level or upon the spinal cord itself.
- This type of condition may occur in the younger population due to injury or it can occur as a result of degeneration and cervical spondylosis.
- When pain is severe and unremitting, associated with clear radicular features, suspect disc impingement.
- Neck pain associated with degenerative disc disease and osteophytes will improve in the majority of people without invasive treatment; although there is certainly a group that will go on to have chronic symptoms.

4. SPINAL STENOSIS

- Cervical stenosis results in myelopathy.
- It usually progresses slowly, and can be quite subtle in the early stages.
- The most common presenting complaints include neck pain, gait difficulties, and hand numbness and clumsiness.
- Loss of bowel and bladder control is uncommon early in the process.
- Occasionally patients will present with acute and profound spinal cord dysfunction after a hyperextension injury.
- More common is a stepwise decline in spinal cord function.
- The typical patient with cervical stenosis **is older than 50 and male**. Men are seen nearly twice as often as women. Myelopathic findings dominate the physical findings.
- Increased reflexes in both the upper and lower extremities with lower extremity spasticity are common. Abnormal reflexes such as **Babinski and Hoffman are also often present**.
- **Lhermitte's sign** (electric, shock-like pain radiating down the spine on neck flexion) is classically described, but occurs in a small minority of patients.
- Complicating the clinical picture is the lower motor neuron findings that can be seen secondary to nerve root compression, such as wasting, fasciculations, and hypoactive reflexes.
- The differential diagnosis **includes multiple sclerosis, syringomyelia, spinal cord tumor, subacute combined degeneration, and normal pressure hydrocephalus**.
- **Surgical decompression** of the cervical spinal cord is recommended in the setting of any signs of myelopathy and significant cervical canal stenosis.
- Deficits acquired are rarely completely corrected by surgery, so most surgeons will tend to offer decompression as early as possible. In patients with significant cervical stenosis without signs or symptoms of myelopathy, operative indications are less clear.

5. MENINGITIS

- Be vigilant for the neck pain of meningitis, which often creates a typical board rigidity of the neck.
- A patient with spontaneous neck pain who cannot kiss their knees is a candidate.
- **Fever will usually be present**, in a very unwell patient, with possible photophobia and headache. However, the time course is acute.

6. PHARYNGITIS

- Neck pain may occur in pharyngitis, particularly when caused by certain viruses.
- As viral pathology is a common pathology in childhood, the neck pain may be over-emphasised, resulting in a misleading diagnosis. It has a peak occurrence in late summer.
- Distinctive clinical syndromes include acute **lymphonodular pharyngitis** caused by coxsackievirus A10, and **hand-foot-and-mouth disease** caused by coxsackievirus A5, 9, 10, and 16, and enterovirus 71.
- A rare but life-threatening cause of pharyngitis in young adults is **Lemierre's syndrome** (thrombophlebitis of the internal jugular vein).
- **Metronidazole and clindamycin** are effective in combination as first line treatment.
- **Peritonsillar abscesses**
 - It is a potent cause of neck pain, but is usually associated with ipsilateral ear pain, and obvious swallowing difficulty.
 - It usually progresses from tonsillitis to cellulitis and ultimately to abscess formation.
- **Ludwig's angina**
 - It is a serious, potentially life-threatening infection of the tissues of the floor of the mouth, usually occurring in adults with concomitant dental infections.
 - The cause is usually a bacterial infection, most often Streptococcal, although other bacteria can also cause this.
 - The route of infection in most cases is from infected lower third molars or from an infection of the gums surrounding the partially erupted lower third molars.

7. INFECTIVE DISCIITIS AND SPINAL OSTEOMYELITIS

- Infective discitis may occur in the immuno-compromised, often an IV drug user.
- It usually results in fever, and may progress to lytic collapse of the vertebral body, with consequent catastrophic **quadraplegia**.
- The onset is usually insidious, making it difficult to diagnose in the ED.
- A distant focus of infection may provide an infective nidus from which bacteria spread by the bloodstream to the spinal column.
- The skin and the genitourinary tract are common antecedent sites, but there may be multiple foci.
- Typically, the organism most likely to infect the spine is **Staphylococcus aureus**; however, in intravenous drug users, **Pseudomonas species** are also a common cause.
- Nonpyogenic osteomyelitis can be caused by **tuberculosis, fungus, yeast, or parasitic organisms**

- Approximately 30-70% of patients with vertebral osteomyelitis have no obvious prior infection.
- **RISK FACTORS FOR DEVELOPING OSTEOMYELITIS INCLUDE:**
 - *Advanced age*
 - *Intravenous drug use*
 - *Congenital immunodepression*
 - *Long-term systemic administration of steroids*
 - *Diabetes mellitus*
 - *Organ transplantation*
 - *Malnutrition*
 - *Cancer*
- Early investigation with xrays may show no defect, but the **ESR and CRP** are likely to be raised, and a **bone scintigraphy** is likely to be abnormal.

8. METASTASES

- Metastatic deposits create a similar radiological appearance to the lytic appearances of infection, and in many cases are completely covert ie there is no known history of carcinoma.

DIAGNOSTICS

- **Plain radiograph**
 - 3 films are taken of the cervical spine: **lateral, AP and open mouth (peg) views.**
 - **Variations in paediatric patients**
 - Relatively large head leading to higher fulcrum of flexion (C2/3)
 - Horizontally aligned facet joints
 - Underdeveloped uncinate process leading to flatter articular surface
 - Anterior wedging of vertebral bodies
 - Cartilaginous synchondrosis at the junction of the odontoid peg and C2 vertebral body
 - Less rigid ligamentous support and weak supportive muscles
- **CT imaging:** If bony lesions are suspected **this is a good investigation.**
 - In spontaneous neck pain, therefore, if spondylitic change, the presence of **metastases or osteomyelitis** is being considered, CT is the preferred investigation.
- **MRI scanning:** Is the imaging modality of choice **in spinal cord disease.**
- **Myelography:** It may help to find the cause of pain not found by an MRI or CT, but it has been largely replaced by the use of CT and MR scans.
- **MANAGEMENT**
 - Ensure adequate pain relief: combination analgesic therapies may be required
 - Be sure to confidently diagnose the cause of spontaneous neck pain
 - Seek a specialist opinion when an unusual case occurs
- **Physiotherapy**
 - The aim of physiotherapy is to reduce pain, improve posture and improve the range of movement.
 - Treatments often include the manual mobilization of joints segmentally in the cervical spine.

CHAPTER 24. RENAL AND UROLOGY IN ED

I. ACUTE KIDNEY INJURY

• INTRODUCTION:

- Acute kidney injury (AKI) is a sudden, potentially reversible, kidney dysfunction with partial or complete loss of glomerular filtration resulting in electrolyte and fluid abnormalities as well as retention of nitrogenous waste products.
- In contrast, Chronic Kidney Disease (CKD) describes loss of kidney function for at least three months.
- While a decrease in Glomerular Filtration Rate (GFR) is used to categorize CKD, an increase in serum creatinine or decrease in urine output is used to characterize AKI.
- The most recent definition of AKI is provided by the Kidney Disease: Improving Global Outcomes (KDIGO) group which reconciled the 2004 RIFLE criteria and the follow-up AKIN update.
- Accordingly, acute kidney injury is defined by any of the following:
 - Increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours; or
 - Increase in serum creatinine by ≥ 1.5 times baseline within seven days; or
 - Urine volume < 0.5 mL/kg/h for six hours

• CLASSIFICATION

- Acute kidney injury is classified into three categories (**Prerenal, Intrinsic, or Postrenal**) based on the physiologic mechanism that prevents production or excretion of urine.
- The excretion of urine requires (1: Prerenal) adequate volume of blood flow with adequate hydrostatic pressure, (2: Prerenal/Intrinsic) ultrafiltration by the glomeruli which is dependent on glomerular structure, hydrostatic capillary pressure and oncotic pressure, (3: Intrinsic) tubular reabsorption and secretion of various solutes and reabsorption of most of the free water, and (4: Postrenal) drainage of the resultant urine via the genitourinary tract extending from the renal pelvis to the urethral meatus.
- A patient may have more than one cause of acute kidney injury simultaneously due to derangements of any of the above four processes.
- Additionally, most patients with previously normal kidneys may not have an elevation in serum markers in the event of an acute injury to one kidney.

• Prerenal

- **Hypovolemia:** commonly due to decreased intake, increased losses, diuretic use, blood loss, third spacing, salt-wasting nephropathy, hypoaldosteronism
- **Hypotension:** shock state, heart failure, antihypertensive use, Addison's disease
- **Renal artery vascular diseases:** renal artery stenosis or fibrosis, thromboembolic events, aortic dissection, aortic aneurysm, NSAID/ARB/ACE inhibitor use, pre-eclampsia, HUS, DIC, traumatic devascularization
- **Other:** hepatorenal disease, aortic cross-clamping, renal vein thrombosis

• Intrinsic

- **Interstitial diseases:**
 - Infection (pyelonephritis, infected stone, abscesses, emphysematous pyelitis),
 - Infiltration (amyloid, myeloma, sarcoid, lymphoma),
 - Autoimmune (SLE),
 - Drug-induced interstitial nephritis, loss of parenchyma due to polycystic disease

- **Glomerular diseases:** post-infectious glomerulonephritis, HSP, SLE, Wegener's, Goodpasture's, membranoproliferative.
- **Nephrotoxins:** may result in interstitial and glomerular injury:
 - **Medications:** NSAIDs, aminoglycosides, radiocontrast, amphotericin, sulfonamides.
 - Heme moieties from hemolysis or rhabdomyolysis,
 - Uric acid, calcium oxalate, amyloid deposits
- **Postrenal acute kidney injury**
 - **Intraluminal obstruction:** calculi, obstructed catheters, urethral strictures, posterior urethral valves, vesicoureteral reflux, failed ureteral stents
 - **Extrinsic compression:** BPH, GU/GYN/GI cancers, pregnancy, ascites, expanding hematoma, penile fractures
 - **Other:** traumatic disruption, neurogenic bladder, spinal cord injury (cauda equina syndrome), anticholinergic and alpha-adrenergic antagonist toxicity, surgical injury.
- **CLUES FOR ACUTE RENAL INJURY**
 - **By etiology:**
 - **Prerenal Acute Kidney Injury** may present with hypotension, tachycardia, shock, peripheral edema, vomiting, diarrhea, acute blood loss, flank or back pain, oliguria or anuria.
 - **Intrinsic Acute Renal Injury** may present with flank and back pain, hematuria, proteinuria, urinary casts and sediments, infectious prodrome, and history or presentation of systemic diseases causing microangiopathy and hemolysis like HUS, TTP, scleroderma, and DIC.
 - **Postrenal Acute Kidney Injury** patients present with obstruction to urine flow associated with a history of renal calculi, urinary urge, failure to void, incontinence, mechanical failure of indwelling catheter, pelvic and flank pain, palpable large urinary bladder, CVA tenderness, and hydronephrosis or hydroureter on imaging studies.
 - **By consequence:**
 - Acute kidney injury may present with a variety signs and symptoms consistent with uremia, electrolyte disturbances, and fluid status.
 - These may include third spacing with shortness of breath, pleural and pericardial effusions, interstitial edema, and ascites.
 - **Hyperkalemia** may present with acutely life-threatening arrhythmias and requires emergent diagnosis and management.
 - **Uremia** may present with uremic pericarditis, effusion and life-threatening pericardial tamponade requiring emergent diagnosis and management.
 - **Acute hypertension** with hypertensive emergency also requires emergent management.
- **ED-FOCUSED WORK-UP**
 - **Labs** – Serum chemistry, CK, BUN/Cr ratio, FeNa, Specific gravity, Microscopic analysis, Urine electrolytes
 - **ECG** – evaluate for changes secondary to electrolyte changes
 - **CXR** – volume status, infection
 - **KUB** – displaced ureteral stents, nephrolithiasis
 - **U/S** – hydronephrosis, hydroureter, bladder distention, flow doppler of the kidney
 - **CT** – nephrolithiasis, abdominal/pelvic masses

COMPLICATIONS OF ACUTE KIDNEY INJURY

- Complications of AKI include:
 - **Biochemical:** metabolic acidosis, hyperkalaemia, and other electrolyte disturbances (sodium, phosphate, and calcium).
 - **Cardiovascular:** pulmonary oedema, hypertension, myocardial depression, arrhythmias, pericarditis.
 - **Gastrointestinal:** GI bleeding, gastric stasis, ileus, anorexia, vomiting.
 - **Haematological:** anaemia, impaired haemostasis, platelet dysfunction.
 - **Neurological:** lethargy, memory impairment, encephalopathy, peripheral neuropathy.

MANAGEMENT OF AKI IN ED

- The main treatment modalities for AKI in the ED are:
 - Fluid resuscitation and monitoring of volume status.
 - Prevention of further injury by stopping nephrotoxic drugs.
 - Urinary catheterization to relieve any urethral or bladder obstruction.
 - Treatment of complications (e.g. hyperkalaemia, pulmonary oedema).
 - Treatment of the precipitant (e.g. sepsis, hypovolaemia).

RENAL REPLACEMENT THERAPY (RRT)

- The main methods of renal replacement therapy (RRT) are:
 - *Intermittent haemodialysis (IHD).*
 - *Peritoneal dialysis (PD).*
 - *Continuous renal replacement therapy (CRRT).*
- Patients requiring long-term renal replacement therapy usually undergo IHD two or three times per week or alternatively PD. These modes are not commonly used in critically ill patients.
- IHD is associated with significant haemodynamic instability in critically ill patients and PD is not capable of removing large volumes of fluid or solute.
- CRRT allows more gradual correction of biochemical abnormalities and removal of fluid, allowing better control of uraemia and clearance of solutes, and avoiding hypotension. CRRT is usually provided in intensive care.
- **Types of CRRT include:**
 - Continuous venovenous haemodialysis (CVVHD)
 - Continuous venovenous haemofiltration (CVVHF)
 - Continuous venovenous haemodiafiltration (CVVHDF)
- Indications for and timing of RRT in AKI is widely debated. The UK Renal Association advice for starting RRT in patients with AKI is that it should be a clinical decision based on the fluid, electrolyte, and metabolic status of the patient.
- **The main ED indications for initiation of RRT are:**
 - Resistant hyperkalaemia ($K^+ > 6.5$).
 - Pulmonary oedema.
 - Refractory metabolic acidosis ($pH < 7.15$).
 - Severe poisoning (e.g. methanol, ethylene glycol, aspirin, theophylline, lithium).
 - Complications of uraemia (e.g. pericarditis).

II. ACUTE RHABDOMYOLYSIS

DEFINITION

- Rhabdomyolysis literally means striated muscle dissolution or disintegration.
- The syndrome is characterised by muscle breakdown and necrosis resulting in leakage of intracellular muscle constituents (Myoglobin, proteins and electrolytes) into the extracellular fluid and circulation.
- Clinically, the most apt definition of rhabdomyolysis is an acute increase in serum concentration of **creatinine kinase (CK)** to greater than five times the upper limit of normal (with myocardial infarction excluded).
- The normal range is between **30–190 IU/litre**. So readings over 200 can be considered as elevated. **Anything over 950 IU/litre** is diagnostic of rhabdomyolysis.

CAUSES:

- Change in medication: statins
- Drug abuse/Alcoholism
- Overexertion
- Genetic disorders
- Heatstroke
- Crush injury
- The most frequent causes of rhabdomyolysis that present to UK emergency departments are **alcohol abuse, muscle overexertion, muscle compression** and the **use of certain medications and illicit drugs**.
- In the USA it is alcohol intoxication that is most commonly associated with prolonged muscle compression and seizures.

CLINICAL MANIFESTATIONS

LOCAL FEATURES	SYSTEMIC FEATURES
<ul style="list-style-type: none"> ◦ Muscle pain ◦ Weakness ◦ Tenderness ◦ Swelling ◦ Bruising 	<ul style="list-style-type: none"> ◦ Dark urine (Tea coloured urine) ◦ Fever ◦ Malaise ◦ Nausea and vomiting ◦ Agitation ◦ Delirium ◦ Anuria

DIAGNOSIS

- The common scenarios that are associated with rhabdomyolysis will be evident from the patient's history or presentation.
- In immobilisation, crush injury and illicit drug use consideration of the diagnosis is obvious. Rhabdomyolysis must also be considered when there is history of recent medication changes, especially statins.
- Remember that in non-traumatic rhabdomyolysis patients may only demonstrate muscle weakness, tenderness or stiffness.
- Paralysis and severe weakness may suggest very extensive myonecrosis or coexistent potassium disturbances that can occur as renal function is impaired.
- Do not dismiss as dehydration a patient who complains of darker than normal urine; **obtain a myoglobin dipstick**.
- The number of rhabdomyolysis patients who develop some degree of renal failure is as great as 50%.

INVESTIGATIONS

- **Blood:** CK, U&E, FBC, calcium & Phosphate, Urate and clotting test
- **Imaging:** MRI, US, CT
- **Urines:** Myoglobinuria

MANAGEMENT OF RHABDOMYOLYSIS IN THE ED

- **Find and Treat the cause**
- **Rehydration**
 - Aggressive intravascular fluid rehydration up to **10 litres of fluid**
 - The sooner this commences the lower the risk of developing renal failure.
 - Ideally rehydration commences pre-hospitally at the same time as extrication.
- **Find and treat complications**
 - Rhabdomyolysis can lead to cardiac arrhythmias as a consequence of **metabolic acidosis and hyperkalaemia**.
 - These disturbances are as important to correct as the arrhythmia itself.
- There are two treatments which are unproven and are considered here, the administration of sodium bicarbonate and the use of mannitol.
- **Administration of sodium bicarbonate**
 - Sodium bicarbonate has been long advocated as a treatment for rhabdomyolysis.
 - The theory was alkalisation of the urine would clear an increasingly acid load delivered to the kidney.
 - There is no evidence to substantiate this. Furthermore, large doses of bicarbonate may worsen the **hypocalcaemia** especially if hypovolaemia is corrected.
 - It is likely that large volume of crystalloid alone will produce a diuresis sufficient to alkalise urine.
- **Use of mannitol**
 - Mannitol has been suggested, and demonstrated in experimental models, to produce a diuresis that protects against renal failure.
 - However robust evidence is lacking from the literature to confirm its efficacy.
 - Mannitol, like furosemide, is a renal vasodilator and osmotic diuretic and both have been used to attempt to initiate diuresis when the patient becomes anuric.
 - Again there is little evidence and retrospective studies suggest there is no additional benefit over fluid hydration.
 - The prognosis in rhabdomyolysis is related to coexistent illness and injury but the renal failure is usually reversible.

III. URINARY RETENTION

OVERVIEW

- Urinary obstruction is a blockage of the flow of urine out of the body.
- It is a common cause of acute and chronic renal failure and may result from a wide variety of pathologic processes, intrinsic and extrinsic to the urinary system.
- **Acute urinary retention:**
 - Comes on quite suddenly and can cause great discomfort or pain. With acute urinary retention, a person cannot urinate at all (even if they have a full bladder) and is a potentially life-threatening medical condition requiring immediate emergency treatment.
- **Chronic urinary retention:**
 - Can be a long-lasting medical condition. Patients with chronic urinary retention can urinate, but they cannot completely empty all of the urine from their bladders.
 - Urinary retention can affect anyone. However, men in their fifties and sixties are more susceptible, primarily because of an enlarged prostate.
 - A woman can also be affected by urinary retention if she suffered from a **Cystocele**, where the bladder starts to sag or moves out of its normal position. It can also be pulled out of position when the lower part of the colon starts to sag — **Rectocele**.

SIGNS AND SYMPTOMS

- **Acute urinary retention symptoms require immediate medical attention:**
 - Inability to urinate
 - Painful, urgent need to urinate
 - Severe pain or discomfort in the lower abdomen
 - Bloating of the lower abdomen
- **Chronic urinary retention symptoms may include**
 - Urinary frequency — urination eight or more times a day
 - Trouble beginning a urine stream
 - Weak or an interrupted urine stream
 - Urgent need to urinate with little success when trying to urinate
 - Feeling the need to urinate after finishing urination
 - Mild and constant discomfort in the lower abdomen and urinary tract
 - Difficulty fully emptying the bladder
 - Urge incontinence/ Inability to feel when bladder is full
 - Increased abdominal pressure
 - Strained efforts to push urine out of the bladder
 - Nocturia (waking up more than two times at night to urinate)

CAUSES

- **Infants and children**
 - **Urethral and bladder outlet obstruction may be associated with the following:**
 - *Urethral atresia*
 - *Phimosis*
 - *Meatal stenosis*
 - *Anterior and posterior urethral valves (males)*
 - *Calculus (Southeast Asia)*
 - *Blood clot*
 - *Neurogenic bladder (meningomyelocele)*
 - *Ureterocele*

○ **Ureteral obstruction may be associated with the following:**

- *Vesicoureteral reflux (female preponderance)*
- *Ureterovesical junction narrowing or obstruction*
- *Ureterocele*
- *Retrocaval ureter*
- *Retroperitoneal tumor*
- *Megaureter - Prune belly syndrome*
- *Blood clot*
- *Ureteropelvic junction narrowing or obstruction*

● **Adults**

○ **Urethral and bladder outlet obstruction may be associated with the following:**

- *Phimosis*
- *Stricture (male preponderance)*
- *Sexually transmitted diseases (STDs), particularly in women with severe genital herpes involving the urethral orifice, occasionally in males with significant prostatitis or purulent urethritis*
- *Trauma*
- *Blood clot*
- *Calculi*
- *Benign prostate hypertrophy (BPH)*
- *Cancer of prostate or bladder*
- *Carcinoma of cervix or colon*
- *Neurogenic bladder:*
 - *Diabetes Mellitus,*
 - *Spinal cord disease,*
 - *Multiple sclerosis,*
 - *Parkinson disease,*
 - *Drugs: anticholinergic drugs, alpha-adrenergic antagonists, calcium channel blockers, opioids, sedative-hypnotics*

○ **Ureteral obstruction may be associated with the following:**

- *Vesicoureteral reflux (female preponderance)*
- *Calculi*
- *Uric acid crystals*
- *Blood clot*
- *Trauma*
- *Papillary necrosis (sickle cell disease, diabetes mellitus, pyelonephritis)*
- *Inflammatory bowel disease*
- *Pregnant uterus*
- *Aortic aneurysm*
- *Carcinoma of ureter, uterus, prostate, bladder, colon, or rectum*
- *Retroperitoneal fibrosis*
- *Idiopathic tumors (cervix, uterus, prostate, colon)*
- *Tuberculosis*
- *Sarcoidosis*
- *Chronic UTI (methysergide, propranolol)*
- *Retroperitoneal lymphoma*
- *Uterine leiomyomata*

- Stricture (tuberculosis, radiation, schistosomiasis, nonsteroidal anti-inflammatory drugs [NSAIDs])
- Accidental surgical ligation
- **Intrarenal obstruction may be associated with the following:**
 - Crystals (uric acid, sulfonamide, acyclovir)
 - Protein casts (multiple myeloma, amyloidosis)

DIAGNOSIS

• Physical examination:

- Palpable kidney or bladder provides direct evidence of a dilated urinary collection system.
- Rectal and/or pelvic examination is essential in determining whether enlargement of pelvic organs (eg, prostate, uterus) is a possible source of urinary obstruction.
- Examination of the external urethra may disclose phimosis or meatal stenosis.

• Lab studies

- Urinalysis and examination of sediment
- Urinary diagnostic indices (eg, sodium, creatinine, osmolality)
- U&E, FBC, CMP

• Imaging studies

- **CT KUB:** Helical CT (especially without contrast) rapidly is replacing kidneys-ureters-bladder (KUB) x-rays as the first step in the radiologic evaluation of the urinary system
- **MRI:** Where available, MRI quickly is becoming the imaging study of choice for urinary obstruction
- **IV pyelography (IVP):** IVP is the procedure of choice for defining the extent and anatomy of obstruction
- **Invasive pyelography:** This modality provides the same information as IVP without depending on renal function and can be used when the risks of IVP are considered too great
- **Ultrasonography:** This is the procedure of choice for determining the presence of hydronephrosis

COMPLICATIONS

• In the longer term, obstruction of the urinary tract may cause:

- Bladder stones
- Atrophy of the detrusor muscle (atonic bladder is an extreme form)
- Hydronephrosis (congestion of the kidneys)
- Hypertrophy of the detrusor muscle (the muscle that squeezes the bladder to empty it during urination)
- Diverticula (formation of pouches) in the bladder wall (which can lead to stones and infection)

TREATMENT

- In acute urinary retention, **urinary catheterization** or **suprapubic cystostomy** relieves the retention.
- In the longer term, treatment depends on the cause.

UNDWELLING URINARY CATHETER

- May be via the urethral (size Ch 14) or supra-pubic route.
- Only make one attempt at urethral catheterisation before calling for help.
- The volume passed within 15 minutes of catheterisation must be recorded.
- Send a C.S.U.
- Check that the foreskin is not left retracted.
- Patients undergoing urethral catheterisation are **not** routinely given prophylactic antibiotics. However, if the dipstick urine is suggestive of infections, consider **80mg Gentamicin IV / IM** prior to instrumentation of the urinary tract.

INDICATIONS FOR CATHETERISATION

- Painful inability to pass urine (acute urinary retention)
- Chronic urinary retention associated with urinary sepsis
- Chronic urinary retention with renal dysfunction i.e. serum creatinine > 200 mmol/l
- Occasionally overflow incontinence due to chronic retention

ASSESSMENT

- Take a good history of previous urinary problems.
- Check U. & E. plus creatinine.
- Record drug history and if episode of retention was related to medication, alcohol consumption or constipation.
- Digital Rectal Examination (DRE).
- If suspicious of UTI commence **Trimethoprim, 200 mgs. bd.**
- Contact urology doctor on call.
- **Do not do a P.S.A.** (retention increases P.S.A. levels).
- As alpha-blockers increase the chance of successful TWOC, provide all patients with a prescription for **Doralese 20mg b.d. PO**, unless contraindicated

COMPLICATIONS OF INDWELLING CATHETERISATION (IDC)

- **Insertion**
 - Malposition
 - Trauma – false passage, urethral stricture (delayed), hemorrhage, balloon inflation in urethra
 - Pain
 - Failure (e.g. meatal, urethral or prostatic stricture – may require SPC or dilation)
- **When in situ**
 - Infection – 100% colonised at 1 week, 5% risk of septic complication per day, 8% bacteremia, 1-3% UTI
 - Paraphimosis
 - Bladder irritation and erosion
 - Hemorrhage post-decompression (if >1 litre bladder)
 - Concretion formation
- **Removal**
 - Traumatic removal (e.g. Balloon not deflated, concretions)
 - Unable to remove (e.g. balloon won't deflate, concretions)
- **OTHER INFORMATION**
 - Administer antibiotics prior to IDC insertion if infection suspected
 - Review ongoing need for IDC daily and monitor for infection

IV. RENAL COLIC

INTRODUCTION

- Renal colic is caused by renal calculi (stones) as they pass through the ureter to the bladder.
- Renal stones may develop and may travel spontaneously through the ureter and into the bladder where they are passed out.
- The pain caused by the passage of stones through the ureter is often compared to the **contractions of childbirth**.
- The pain is due to ureteric muscle contracting in an attempt to shift the stone.

STONE COMPOSITION

- **Calcium oxalate** and / or **phosphate: 60-80%**
- **Struvite 10-15%** and approximately **Cystine 1%** and **Uric acid 1%**.
- Occasionally they are made of **xanthine, indinavir** and **triamterene**.

AETIOLOGY

- **Low urine volume** is the most common factor in patients who tend to form stones and is easily remedied by increasing fluid intake sufficient to produce a urine output of 3 litres per day.
- Usually, a causative factor is not identified.
- The lifetime risk of contracting renal colic is approximately **10%**; Peak age for presentation is **30 years** and **men are twice as likely as women to acquire renal colic**.
- The recurrence rate is approximately **50% over a 10-year period**.
- A positive family history triples the risk of acquiring renal colic.
- Caucasians are more commonly affected than either Asian or Black people.
- Predisposition can also be biochemical or anatomical

CLINICAL ASSESSMENT

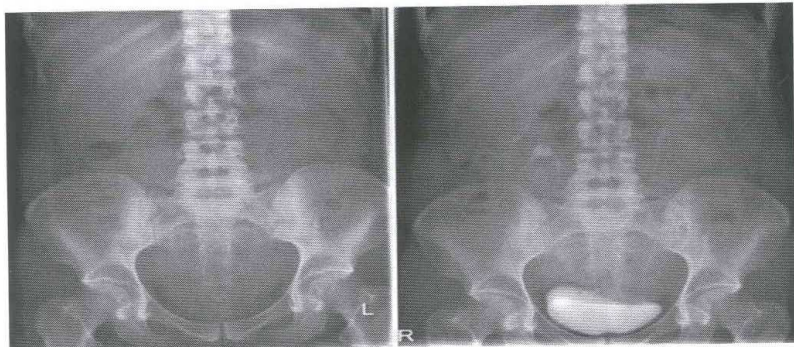
- **Classical presentation**
 - **Sudden onset of unilateral loin to groin or renal pain**, is writhing in agony or restless, and is **nauseated**. Pain is classically colicky in nature and radiates to the scrotum and tip of the penis or labia majora.
 - There may be intervals between episodes of pain, so it cannot be assumed that cessation of pain means that the stone has been passed and the episode ended.
- **Other presentations**
 - Large stones in the renal pelvis will present with **haematuria, infection** or **decreased renal function** rather than acute renal colic.
 - These may be **staghorn** shaped and usually consist of struvite or cystine.
 - **Pyuria** may be evident, but this is usually a result of ureteral irritation rather than a sign of infection.

DIFFERENTIAL DIAGNOSIS

- | | |
|---|---|
| <ul style="list-style-type: none"> • Pancreatitis • Appendicitis • Ovarian pathology and torsion • Pelvic inflammatory disease • Pregnancy • Renal infarct • Aortic aneurysm | <ul style="list-style-type: none"> • Cholecystitis • Renal carcinoma, pyelonephritis • Incarcerated hernia, e.g. abdominal and lumbar • Diverticular disease • Pneumonia |
|---|---|

INVESTIGATIONS

- Patients with renal colic that has seemed to resolve should be imaged, to eliminate differential diagnoses.
- **Urinalysis**
 - **Dipstick urine testing**
 - **Microscopic haematuria** will be present in 90% of cases but it will be absent in up to 10% of cases; therefore, if the clinical presentation is suggestive, these patients should progress to further investigation.
 - **Pyuria** is often present but this is often as a result of ureteral irritation rather than infection.
 - **Microscopy and culture** if infection is suspected from the history or examination.
- **24-hour urine collection**
 - Recurrent stone formers should have a 24-hour urine collection for: Urine volume, Calcium/ Oxalate/Uric acid/Citrate, Sodium and Creatinine
 - Specific therapy will be indicated if abnormalities are found in the course of this screening, e.g. Allopurinol for uric acid stones, and measures required to increase calcium excretion in hypercalcaemia, such as thiazide diuretics.
- **Blood tests: FBC, U&E, Amylase, Phosphate, Urate, Bicarbonate and Calcium**
- Intravenous (IV) access should be obtained at the same time as blood samples are taken for fluid, analgesia or antiemetic administration
- **Plain KUB and Abdominal radiography**
 - Abdominal radiography, including the kidney ureter and bladder (KUB) radiograph, is commonly the first step in the work up of abdominal pain (see figures).
 - The KUB shows no evidence of renal tract calcification and following contrast (right) there is prompt excretion through the upper tracts. These are normal films.



- **Intravenous urography (IVU)**
 - **Disadvantages include:**
 - High degree of expertise needed to interpret the IVU (main disadvantage)
 - The need for contrast administration
 - The radiation dose (although less than CT)
 - The delay in obtaining relevant information about the possible site of obstruction
 - The difficulty in delivering emergency patients to x-ray in an ED
 - Certain patients should avoid IVU: Patients with **renal failure, contrast allergy, pregnant women and diabetics taking metformin**.

- **CT-KUB:** Imaging modality of choice



- CT is rapid, avoids contrast and facilitates alternative diagnostics, especially red flag conditions
- **Ultrasound scanning**
 - Ultrasound has a lower sensitivity (24-77%) when compared to CT (96%).
 - Specificity when both techniques are used approaches 100%.
 - Ultrasound is often used as the **first line imaging technique in pregnant women and children where radiation is best avoided.**
 - Calculi as small as 0.5 mm can be visualised but sensitivity increases with increasing stone size (see Figure).
 - Visualisation of ureteral jets within the bladder lumen on ultrasound disappears with obstruction and may indicate the presence of ureteral obstruction.
 - Twinkling artifact on Doppler may aid the diagnosis of smaller stones.



- **Imaging in pregnancy**
 - Renal colic is no more common in pregnancy.
 - Whilst 80% of calculi pass spontaneously, favouring conservative management, the diagnosis can present problems as hydronephrosis of pregnancy can make diagnosis of associated obstructing calculus difficult.
 - **Endovaginal USS** images can help with visualisation in distal ureteric and ureterovesical stones.
 - **Unenhanced CT** may be indicated in later pregnancy as the exposures required to facilitate IVU exceed the radiation dose.

ED MANAGEMENT OF RENAL COLIC

- Often stones will pass spontaneously.
- This depends on the size and location of the stone.
- 90% of small stones of 5mm or less pass spontaneously.
- On average the spontaneous passage rate of stones of all sizes is approximately:
 - 25% in the proximal ureter
 - 45% in the mid ureter
 - 70% in the distal ureter
- Most urologists advise sieving urine to facilitate chemical analysis of retrieved fragments, especially in the case of recurrent stone formers.
- Since the majority of renal stones pass spontaneously, management in the ED should be directed to rapid diagnosis and pain relief with referral to urology indicated for developing complications.
- **Analgesia**
 - **Opioids and NSAIDs:** have been used in renal colic and both appear to be effective.
 - Ureteric inflammation may be reduced in NSAIDs administration facilitating stone passage

ADMISSION CRITERIA FOR A PATIENT WITH RENAL COLIC

- *Failure of pain control*
- *Large stone: >6mm unlikely to pass*
- *Solitary kidney or transplanted kidney*
- *Bilateral obstruction*
- *Presence of infections*
- *Impaired Renal function*
- **When is it safe to discharge a patient?**
 - It is only safe to discharge a patient when **pain is under control**.
 - Brief ceasing of pain may occur **during stone passage**, and you must have ruled out differential diagnosis so imaging is always recommended.
 - It is also only safe to discharge patients if there are **no complications** such as **bilateral blockage, single kidney, anuria or signs of infection**. These complications warrant hospital admission.
- Only two drug classes are currently considered to be effective as medical expulsive therapy (MET), **calcium channel blockers and alpha-blockers**.
- **If septic:**
 - The presence of pyrexia mandates urgent full blood count, blood and urine cultures, creatinine and lactate levels should also be obtained.
 - Resuscitation using surviving sepsis guidelines in a High Dependency Unit environment is advised. Antibiotic options include **Gentamicin and Ceftazidime intravenously**.
- **If hydronephrosis**
 - **Urgent nephrostomy** and this is usually performed by a radiologist
- **Patients with stones causing significant ureteric obstruction and pain**
 - **Stenting** pending definitive treatment.
 - Correct insertion is evidenced by x-ray confirmation of the proximal coil in the renal pelvis and distal coil in the bladder. In the 20% of cases where placement fails, then nephrostomy is required. Stents have the potential to cause distressing symptoms and impair quality of life and many urologists are attempting to decrease usage.

V. TESTICULAR SWELLING

• SCROTAL SWELLING: PAINLESS VS PAINFUL

PAINFUL	PAINLESS
• Testicular torsion	• Testicular tumour
• Torsion of Appendix testis	• Hydrocoele
• Trauma	• Varicocele
• Epididymitis	• Spermatocoele
• Epididymal cyst	

1. EPIDIDYMITIS

BACKGROUND

- Acute epididymitis is an infection of the epididymis.

CLINICAL

- Pain in the scrotum usually develops quite quickly.
- The patient may notice a rapid swelling of the affected hemiscrotum.
- Irritative voiding symptoms and fever may also be present.
- On exam, the hemiscrotum is usually visibly enlarged and the overlying skin reddened.
- The affected epididymis is quite tender. At first the indurated epididymis may be distinguishable from the testicle but as the inflammatory process continues the epididymis and testicle become one inflammatory mass. A reactive hydrocoele may also develop. **Rectal exam** should be done to rule out prostatitis as the source of infection.

INVESTIGATIONS

- The patient may have an elevated **white count and positive urinalysis** but this is not always the case. Urine should be routinely sent for **culture and sensitivity**.

ED MANAGEMENT

- The diagnosis is often difficult to make because of the similar presentation of testicular torsion.
- **If there is any possibility that torsion exists then a urologist should be consulted.**
- **A Doppler ultrasound or Testicular Flow Scan** can sometimes be helpful in distinguishing the two conditions but imaging studies should not be done if they will delay surgical treatment. In a young man a **sexual transmitted organism** is the most likely cause.

2. TESTICULAR TORSION

BACKGROUND

- Torsion occurs when the spermatic cord twists and compromises the blood supply to the testicle.
- It occurs most commonly in the adolescent age group and can follow minor trauma.
- The patient typically develops acute onset severe unilateral testicular pain.
- The pain may also radiate to the lower abdominal with nausea and vomiting.
- **Examination reveals that the:**
 - *Scrotal skin edematous and erythematous*
 - *Testis too tender to touch*
 - *Testis lies high in scrotum (Deming's sign)*
 - *Opposite testis lies horizontally (Angel's sign)*
 - *Pain not relieved by elevating testis (Negative Prehn's sign)*
 - *Absence of cremasteric reflex*

- Scrotal elevation relieves pain in epididymo-orchitis but not in torsion (**Prehn's sign**).
- This sign may be difficult to test reliably in children.
- **The cremasteric reflex** has 100% sensitivity and 66% specificity (the cremasteric reflex can be absent in neonates and in people with neurological disorders).
- **The cremasteric reflex (L1/L2 spinal nerves)** - gentle pinching or stroking of the inner thigh while observing the scrotal contents. The normal response, owing to shared innervations, is for the cremasteric muscle to contract, resulting in elevation of the ipsilateral testicle.

ED MANAGEMENT OF TESTICULAR TORSION

- This requires an **urgent urology consult**.
- If the diagnosis of torsion is suspected **surgical exploration** is necessary.
- The spermatic cord must be untorted **within 6 hours** if the testicle is to be saved.
- Whether or not the testicle has undergone torsion it should be sutured down to the scrotal skin to preclude any subsequent torsion and any uncertainty over the diagnosis should the pain recur. Once the testicle has been surgically tacked down it should never twist again. The opposite testicle should also be sewn down since the anatomic abnormality that caused torsion on one side may be present bilaterally.
- If, however, the testicle does not appear viable intra operatively **it should be removed**.
- It has been shown that leaving a non-viable testicle in situ **will significantly decrease the patient's future fertility**. This is most likely due to an **autoimmune phenomenon** which occurs as the body is exposed for the first time to its own sperm.

3. TORSION OF TESTICULAR APPENDAGE

- Torsion of the testicular appendages can also occur. **The appendix testis** is by far the most common of the appendages to twist. It presents as acute onset unilateral scrotal pain in the adolescent. Usually a **tender pea-sized nodule** can be palpated at the upper pole of the ipsilateral testis. If the appendix testis has infarcted, a **small blue dot** can sometimes be seen through the scrotal skin "**BLUE DOT SIGN**".
- Surgical exploration is usually required.
- If an infarcted appendix is found it should simply be excised.
- However, in the acute setting, differentiating testicular torsion from torsion of the appendix is often impossible, and **scrotal exploration** should be performed whenever the diagnosis is uncertain.

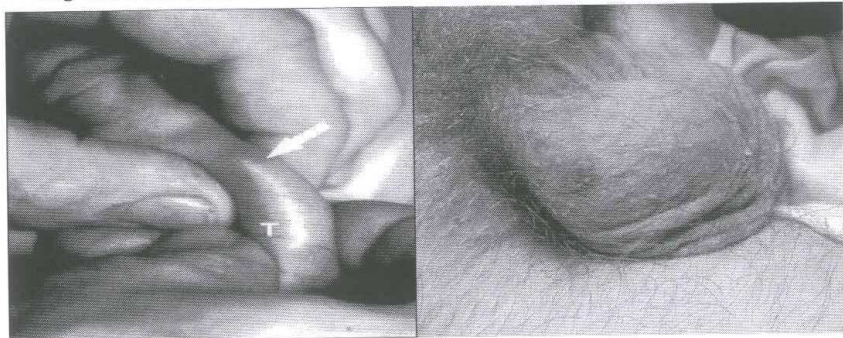


Figure 1.24.1. Blue dot sign

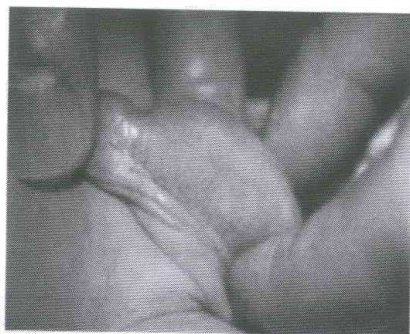


Figure 1.24.2. Blue Dot Sign (Torsion of appendix testis)

- A classic **"blue-dot sign"** (ie, a tender nodule with blue discoloration on the upper pole of the testis) may be seen; *this finding on the upper scrotum is a typical finding in torsion of the appendix testis.*

DIFFERENTIAL DX

- Problems to be considered in the differential diagnosis of testicular torsion include the following:
 - Torsion of testicular or epididymal appendage
 - Epididymitis, orchitis, epididymo-orchitis
 - Hydrocele
 - Testis tumor
 - Idiopathic scrotal edema
 - Idiopathic testicular infarction
 - Traumatic rupture
 - Traumatic hematoma

4. TESTICULAR TUMOUR

- Testicular tumours represent only 1-2% of all tumours in men but are the second most common malignancy in males between the ages of 20 and 35 years.
- Almost all are germ cell tumours and are classified as either **seminomas, nonseminomas or a mixture of the two**. They classically present as **painless unilateral scrotal swellings**.
- On physical exam there is a firm, non tender, intra testicular mass that does not transilluminate.
- **A scrotal ultrasound** may be helpful if there is any question about the diagnosis.
- If a testicular tumour is suspected a prompt urology consult is necessary as these can be rapidly growing tumours. The diagnosis must be made **pathologically**.
- Once the diagnosis is confirmed the disease must be staged. Testicular tumours usually spread in an orderly fashion first seeding the retroperitoneal lymph nodes.
- **A CT scan of the abdomen and pelvis** can detect lymph nodes greater than 2 cm.
- **A CXR** is also needed to rule out more distant metastases.
- **Tumour markers** including **alpha-fetoprotein** and **beta-HCG** can also be helpful to detect residual tumour mass.
- **Urology referral:** Treatment options differ depending on whether the primary tumour is a seminoma or a nonseminoma, with mixed tumours being treated as nonseminomas.

VI. BALANITIS & POSTHITIS

- **Balanitis** is inflammation of the glans penis, **Posthitis** is inflammation of the prepuce, and **Balanoposthitis** is inflammation of both.
- Balanitis usually leads to Posthitis except in circumcised patients.
- Inflammation of the head of the penis has both infectious and noninfectious causes. Often, no cause can be found.

CAUSES OF PENILE INFLAMMATION

CATEGORY	EXAMPLES
<ul style="list-style-type: none"> • Infectious 	<ul style="list-style-type: none"> • Candidiasis • Chancroid • Chlamydial urethritis • Gonococcal urethritis • Herpes simplex virus infection • Molluscum contagiosum • Scabies • Syphilis, primary or secondary • Trichomoniasis
<ul style="list-style-type: none"> • Noninfectious 	<ul style="list-style-type: none"> • Balanitis xerotica obliterans • Contact dermatitis • Fixed drug eruptions • Lichen planus • Lichen simplex chronicus • Psoriasis • Reactive arthritis • Seborrheic dermatitis

RISK FACTORS

- Balanoposthitis is predisposed to by:
 - Diabetes mellitus
 - Phimosis (tight, nonretractable prepuce)
- Phimosis interferes with adequate hygiene. Subpreputial secretions may become infected with anaerobic bacteria, resulting in inflammation.
- **Chronic balanoposthitis** increases the risk of:
 - Balanitis xerotica obliterans
 - Phimosis
 - Paraphimosis
 - Cancer

SYMPTOMS AND SIGNS

- Pain, irritation, and a subpreputial discharge often occur 2 or 3 days after sexual intercourse.
- Phimosis, superficial ulcerations, and inguinal adenopathy may follow.

DIAGNOSIS

- Clinical evaluation and selective testing
- History should include investigation of latex condom use.

- The skin should be examined for lesions that suggest a dermatosis capable of genital involvement.
- Patients should be tested for both infectious and noninfectious causes, especially **candidiasis**.
- A **swab** may be taken if the diagnosis is uncertain.
- Blood should be tested for **glucose**.

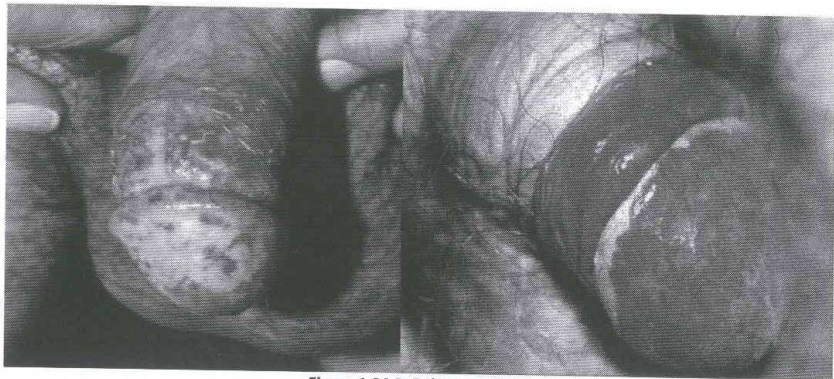


Figure 1.24.3. Balanoposthitis

TREATMENT

- **Good hygiene** and gentle cleaning of the area
- Treatment of specific causes (**Clotrimazole** if candidal infection is suspected)
- Sometimes subpreputial irrigation
- Sometimes circumcision

CHAPTER 25. PAIN MANAGEMENT IN ADULT

PAIN ASSESSMENT

- Pain assessment forms an integral part of the National Triage Scale.
- Multiple assessment tools are in use.
- The better-known scales have not been validated in the context of an ED environment but are nevertheless satisfactory for the purpose of pain assessment and management.
- The recording of pain scores is often suboptimal.
- The experience of the member of staff triaging will help in estimating the severity of the pain.
- The literature suggests that assessment of pain in the ED is often not as good as it could be which is particularly concerning since pain is often the reason for attending, patient assessment is improved by giving adequate analgesia, painful or uncomfortable procedures may be undertaken in the ED and there are clear physiological benefits to providing adequate analgesia.

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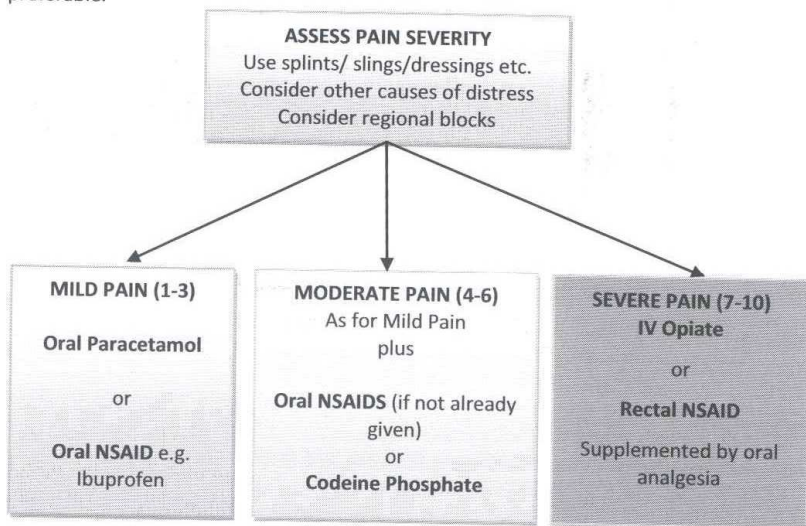
ASSESSMENT OF ACUTE PAIN IN THE ED

Pain score	No pain 0	Mild Pain 1-3	Moderate Pain 4-6	Severe Pain 7-10
Suggested route & Type of Analgesia	No Action	Oral analgesia	Oral analgesia	IV Opiates or PR NSAID
Initial assessment	Within 20 min of arrival	Within 20 min of arrival	Within 20 min of arrival	Within 20 min of arrival
Re-evaluation	Within 60 min of initial assessment	Within 60 min of analgesia	Within 60 min of analgesia	Within 30 min of analgesia

- Using this method of pain scoring it should be possible to adequately assess into one of four categories and treat pain appropriately.
- Once the category has been established, appropriate analgesia may be prescribed according to the flow chart.
- In all cases it is important to think of using other non-pharmacological techniques to achieve analgesia, which may include measures such as **applying a dressing or immobilising a limb** etc.
- Following reassessment if analgesia is still found to be inadequate, stronger / increased dose of analgesics should be used along with the use of nonpharmacological measures.
- It is important to re-assess the pain control within **30-60 minutes** in severe and moderate pain.

HOW TO MANAGE PAIN IN THE ED

- **Patients in severe pain**
 - Should be transferred to an area where they can receive appropriate **intravenous or rectal analgesia within 20 minutes of arrival**. Should have the effectiveness of analgesia re-evaluated **within 30 minutes of receiving the first dose of analgesia**.
- **Patients in moderate pain**
 - Should be offered **oral analgesia at triage / assessment**. Should have the effectiveness of analgesia re-evaluated **within 60 minutes of the first dose of analgesia**.
- **Documentation of analgesia** is essential and departments are encouraged to formalise pain recording in the same manner as the regular documentation of vital signs.
- The guidance in this document is primarily aimed at ensuring patients get appropriate and adequate analgesia in a timely fashion.
- When patients first present to the ED the diagnosis may be unclear and it is important that the lack of diagnosis does not delay administration of appropriate analgesia.
- Emergency physicians are sometimes placed in the very difficult position of having to decide whether a patient's pain is genuine or not (i.e. is the patient displaying 'drug seeking' behaviour?).
- Careful decision making is required to balance the embarrassment of 'being tricked' by a drug seeker as opposed to denying a patient with genuine pain appropriate and adequate analgesia. Being '**wise after the event**' and instituting appropriate measures is likely to be preferable.



• WHEN PRESCRIBING FOR THE ELDERLY

- It is worth remembering that **Paracetamol** (including intravenous) is a safe first line treatment with a good safety profile.

- **NSAIDs** should be used with caution and at the lowest possible dose in older adults in view of gastrointestinal, renal and cardiovascular side effects as well as drug-drug interactions and the effects on other co-morbidities.
- **Opiate medication** in the elderly, an appropriate dose reduction should be used as well as anticipating any other drug interactions; particularly those acting on the central nervous system which may increase the likelihood of respiratory depression.
- **WHEN PRESCRIBING IN PREGNANCY**
 - The general rule is try to avoid any medication, however this is not always practical.
 - **Paracetamol** is considered safe in all three trimesters,
 - **Ibuprofen** is best avoided and can only be used **during the second trimester** (if essential).
 - **Morphine and codeine** can be used in all three trimesters if necessary but should be avoided during delivery.

DRUG NOTES

1. PARACETAMOL

- Available as oral, rectal and intravenous preparations.
- **The standard oral** and IV dose for adults is **1gram qds** however when administering the IV preparation the dose must be adjusted for those patients weighing less than 50Kg (adults 40-49kg 750mg qds, 35-39kg 500mg qds).
- **The IV route** is particularly useful when patients need to be kept nil by mouth and rapid mild-moderate analgesia is required.
- **The rectal preparation** is probably best avoided due to variable and slow absorption in adults. Before prescribing paracetamol, inquiry must be made regarding previous paracetamol use (including preparations such as co-codamol and OTC preparations e.g. cold relief powders as well as paramedic use prior to arrival in the ED).

2. NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

- Available as oral, rectal, intravenous and intra-muscular preparations (although it should be noted **IM diclofenac** has been associated with sterile abscesses following IM use).
- **Ibuprofen 400mg PO tds**; fewer side effects than other NSAIDs, good analgesic but relatively weak anti-inflammatory properties.
- **Naproxen 500mg PO initially then 250mg every 6-8hrs** in acute musculoskeletal disorders; stronger anti-inflammatory properties than ibuprofen but with relatively fewer side-effects compared to other NSAIDs.
- **Diclofenac 50mg PO tds, 100mg PR**; particularly useful for the treatment of renal colic pain via the rectal route however in recent years' concern has been raised regarding increased risk of thrombotic events (incl. MI) and *Clostridium difficile* and it is **contra-indicated in IHD, PVD, CVD and heart failure**.
- Avoid NSAIDs in **asthmatics** who are known to get worsening bronchospasm with NSAIDs, also avoid in patients with previous or **known peptic ulcer disease**.
- NSAIDs should be **used with caution in the elderly** (risk of peptic ulcer disease) and **women who are experiencing fertility issues**.
- It should also be **avoided in pregnancy**, particularly during the third trimester.

3. OPIATES

- **Codeine Phosphate** is available as oral and IM preparations, **30-60mg qds** are typical adult doses however consider lower doses in the elderly.
- Codeine prescribed in combination with paracetamol is significantly more effective than codeine when prescribed alone.
- **Morphine** is available as oral, intravenous and intra-muscular preparations (due to its relatively slow onset of action the oral preparation is not recommended for acute pain control in the ED, unless the patient is already taking the drug in which case this might be a reasonable alternative).
- **Morphine 0.1-0.2mg/kg IV** is a typical adult dose, however a titrated dose to provide the desired response is recommended; consider lower doses in the elderly. Use with caution if **risk of depression of airway, breathing or circulation**.
- The routine **prescription of an anti-emetic with an opiate is not recommended**, and only required if patient is already experiencing nausea / vomiting.
- It should be noted that the use of opioids in abdominal pain does not hinder the diagnostic process.

4. ENTONOX

- **Entonox, a 50% mixture of nitrous oxide and oxygen**, is very useful for short term relief of severe pain and for performing short lasting uncomfortable procedures.
- It should not be viewed as a definitive analgesic and EDs need mechanisms in place to ensure rapid assessment and institution of appropriate analgesia when paramedics bring patients to the ED who are using Entonox as their sole source of analgesia.
- **Entonox should be avoided in patients with:**
 - *Head injuries,*
 - *Chest injuries,*
 - *Suspected bowel obstruction,*
 - *Middle Ear disease,*
 - *Early pregnancy and*
 - *B12 or folate deficiency.*

CHAPTER 26. PALPITATIONS

I. TACHYCARDIA



Resuscitation Council (UK)

GUIDELINES
2015

ADULT TACHYCARDIA (WITH PULSE) ALGORITHM

Assess using ABCDE approach

- Monitor SPO₂ and Give O₂ if hypoxic
- Monitor ECG and BP, and record 12-lead ECG
- Obtain IV access
- Identify and treat causes (electrolyte abnormalities)

Synchronized DC shock
Up to 3 attempts

YES:
UNSTABLE

Seek expert Help

- Amiodarone 300mg IV over 10min
- Repeat shock
- Then amiodarone 900mg X 24hr

Adverse features?

- Shock
- Myocardial Ischemia
- Syncope
- Heart Failure

NO: STABLE

Is QRS < 0.12sec?

NO: BCT

YES: NCT

Broad Complex Tachycardia
Is QRS Regular?

Irregular

Regular

Seek expert Help

Possible causes:

- AF with Bundle Branch Block: treat as for narrow complex
- Pre-excited AF: treat with Amiodarone
- Polymorphic VT (Torsade de pointes): Mg 2g IVI

- If VT: Amiodarone 300mg IV over 20-60min then 900mg X 24hr

- If known to be SVT with Bundle Branch Block Treat as regular NCT

Narrow Complex Tachycardia
Is Rhythm Regular?

Irregular

Regular

Probable AF

- Control rate with β -blockers and Diltiazem
- If in Heart failure, consider Digoxin and Amiodarone
- Assess thrombolytic risk & consider anticoagulation

- Vagal Manoeuvres
- Adenosine 6mg IV Bolus
- If no effect: further 12mg
- If no effect: further 12mg
- If adenosine CI or failed: verapamil 2.5-5mg IV/2min
- Monitor ECG continuously

YES

Sinus rhythm achieved?

NO

Seek expert Help

Possible A flutter:
Control Rate (e.g. B-Blockers)

Probable Re-entry Paroxysmal SVT

Record 12-lead ECG in sinus rhythm
If SVT recurs treat again and consider anti-arrhythmic prophylaxis

1. BROAD COMPLEX TACHYCARDIA

1.1. IRREGULAR BROAD COMPLEX TACHYCARDIAS

- The 3 causes are:
 - Atrial fibrillation with a bundle branch block
 - Pre-excited atrial fibrillation
 - Torsades de pointes

*Any irregular and sustained broad complex tachycardia should be assumed to be **Atrial Fibrillation with either pre-existing Bundle Branch Block or Aberrant Conduction***

- It is rare for such a rhythm to be ventricular in origin. Although polymorphic VT is irregular, it is rarely sustained.
- **Atrial fibrillation** may occasionally masquerade as polymorphic VT when it is in the presence of pre-excitation.

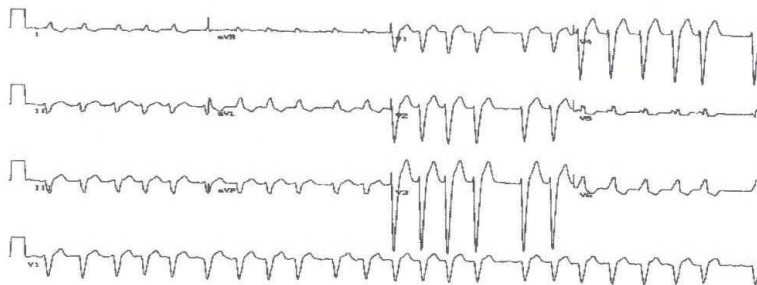


Figure 1.26.1. Atrial fibrillation and LBBB

- The atrial fibrillation gives rise to an irregular rhythm and the variable conduction down the accessory pathway gives rise to QRS complexes which do change in morphology due to the presence or absence of **Delta waves** giving a similar appearance to Torsades de pointes.

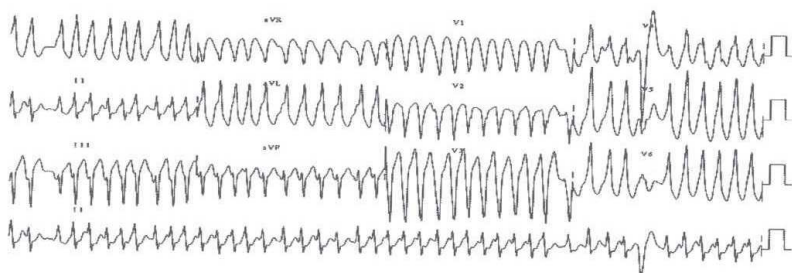


Figure 1.26.2. Wolff Parkinson White and atrial fibrillation

- **Treatment of AF:**
 - Once atrial fibrillation is identified as the underlying arrhythmia, it should be treated as such the treatment of atrial fibrillation is discussed in detail in the relevant module.

1.2. REGULAR BROAD-COMPLEX TACHYCARDIA**a. BCT of Ventricular origin****A. MONOMORPHIC VENTRICULAR TACHYCARDIA**

- Monomorphic refers to a VT where each and every QRS complex is the same shape and size as the next.
- This form of tachycardia frequently occurs after **myocardial infarction** when it is a sign of extensive damage.
- **The cardinal features of monomorphic VT are:**
 - QRS rate is 120-250 beats/min
 - QRS rate is regular
 - QRS configuration is constant
 - QRS configuration is different from that in sinus rhythm
 - The QRS complexes are abnormally wide ($>0.12s$)

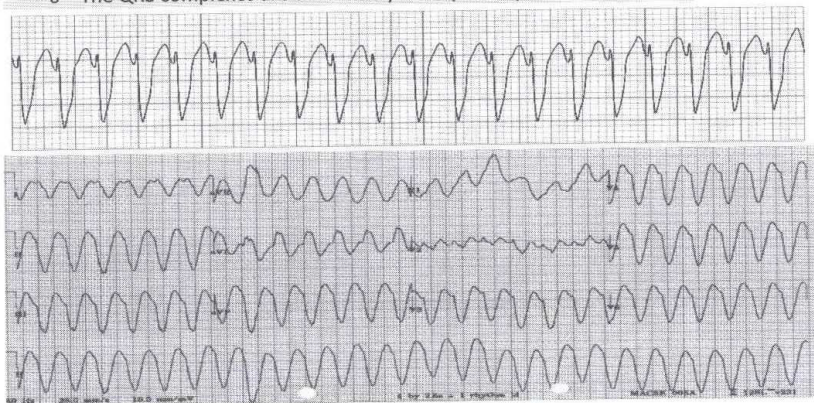


Figure 1.26.3. Monomorphic VT

• TREATMENT OF MONOMORPHIC VT

- **If compromised:** DC cardioversion.
 - Synchronised DC cardioversion at **200 joules** (monophasic) or **100 joules (biphasic)**.
 - If unsuccessful repeat the cardioversion up to a **maximum of 3 attempts** before giving **amiodarone**.
 - Changing the paddle position may be helpful in resistant cases.
- **If stable:**
 - **IV Amiodarone** (in a dose of 5mg/kg up to a maximum of 300mgs) administered over 20-60 minutes is the treatment of choice. If unsuccessful, **DC cardioversion** should be considered. However, **amiodarone** is poorly effective in the treatment of acute VT.
 - **Sotalol** appears more effective in the treatment of stable VT (compared with lignocaine, which was the ALS recommendation at that time).
 - **Procainamide** has class IIa evidence supporting its usage in this situation but is slow to work.
- **DC cardioversion** is reasonable as first-line treatment of stable VT.
- **Correction of any underlying abnormalities** that might be precipitating the arrhythmia (eg. hypo/hyperkalaemia and hypomagnesaemia) is also required.

B. POLYMORPHIC VENTRICULAR TACHYCARDIA

- o Polymorphic refers to the fact that the shape and size of each QRS complex varies from the preceding one.
- **The Cardinal Features of Polymorphic VT:**
 - o QRS rate is rapid (200-250 beats/min)
 - o Amplitude of the QRS complexes change in a sinusoidal fashion
 - o Usually self limiting but may be recurrent
 - o Between episodes the QT interval is prolonged

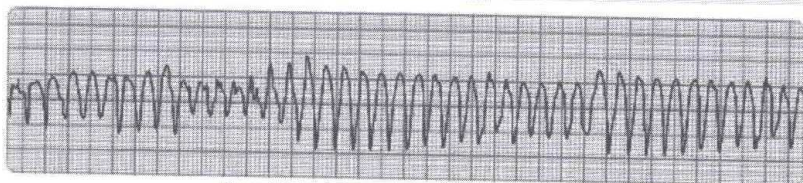


Figure 1.26.4. Polymorphic Ventricular Tachycardia

- **TREATMENT OF POLYMORPHIC VT:**

- o Polymorphic VT that develops in association with an **Acute Coronary Syndrome** requires immediate **DC cardioversion**, as it frequently degenerates into ventricular fibrillation.
- o Those patients with **Torsades de pointes** who are stable require **IV Magnesium** and correction of underlying abnormalities (eg. hypokalaemia, hypoxia, etc.) where appropriate.

C. FASCICULAR TACHYCARDIA

- **The Cardinal Features of Fascicular Tachycardia: RBBB+LAD**

- o Right bundle branch block pattern
- o Left axis deviation
- o QRS duration 110 to 140 mseconds

- Fascicular tachycardia is uncommon.

- **Treatment of Fascicular Tachycardia:**

- o Fascicular tachycardia should be treated in the same way as monomorphic tachycardia.
- o It is important to identify this arrhythmia correctly so that it is not treated as a supraventricular tachycardia: **the administration of verapamil could be dangerous in this setting.**

D. RIGHT VENTRICULAR OUTFLOW TACHYCARDIA/RVOT

- **Cardinal features of RVOT: LBBB+RAD**

- o Left bundle branch block
- o Right axis deviation

- **Treatment of RVOT:**

- o Right ventricular outflow tachycardia usually responds to **adenosine, a beta-blocker or a calcium antagonist.**

b. BCT of Supraventricular origin**DIFFERENCE BETWEEN VT AND SVT WITH BBB**

1. If the patient is >50 and/or has a **history of structural or ischaemic heart disease**, assume the rhythm is VT. If there is any doubt whatsoever, treat a regular broad complex tachycardia as VT.

2. a. The following are suggestive of VT:

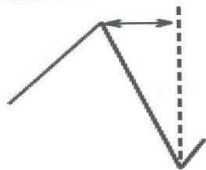
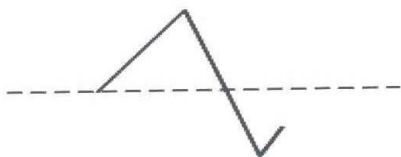
- Dissociated P waves
- Fusion/Capture beats
- A bizarre axis
- QRS >140 msec
- Concordance of the QRS complexes in the chest leads

2. b. Features suggestive of BCT of supraventricular origin:

- Age < 35
- Rate ≈ 150 beats/minute
- Rate >200 beats/minute and patient asymptomatic
- QRS Duration < 140 msec
- Axis normal
- Absence of independent atrial activity or concordance

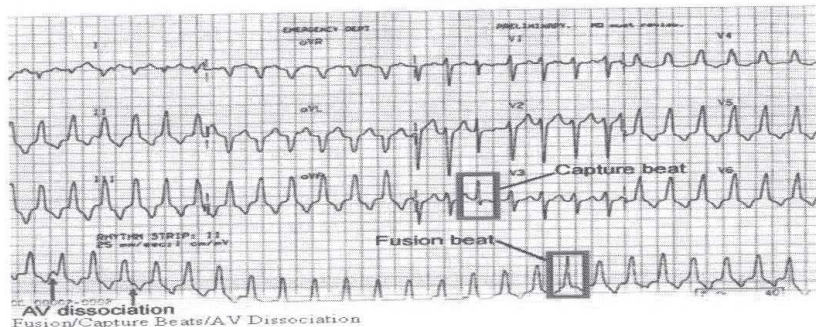
3. Brugada Criteria

- The following should be noted:

RS wave**RS interval**

1. Is there an absence of RS complexes in all the chest leads?

2. Is the R-S interval (interval between the tip of the R wave and the lowest part of the S wave) > 100 ms in any V lead?



3. Are there capture beats, fusion beats, or evidence of AV dissociation?

4. Does the morphology of the QRS complex in leads V1/ V6 suggest VT?

Morphologic Criteria suggestive of VT

1. LBBB morphology

• V1:

- R wave > 30 msec wide
- RS wave > 60 msec wide

• V6:

- QR wave
- QS wave

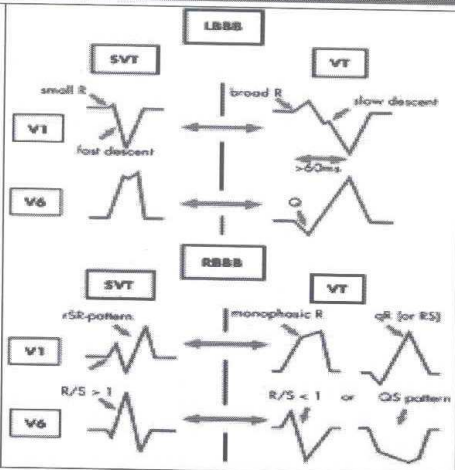
2. RBBB morphology

• V1:

- Monophasic R wave
- QR wave
- RS wave

• V6:

- Monophasic R wave
- QR wave
- R wave smaller than the S wave



- If the answer to **any** of these questions is **YES**, then the diagnosis is VT.
- If the answer to **all** of these questions is **NO**, then the diagnosis is SVT with a bundle branch block.
- **TREATMENT:**
 - **Vagal manoeuvres and Adenosine** (a short acting purine) may be used diagnostically (to help identify BCT which is supraventricular in origin) and therapeutically (to terminate the arrhythmia).
 - Detailed management of supraventricular tachycardia is discussed in a separate module.

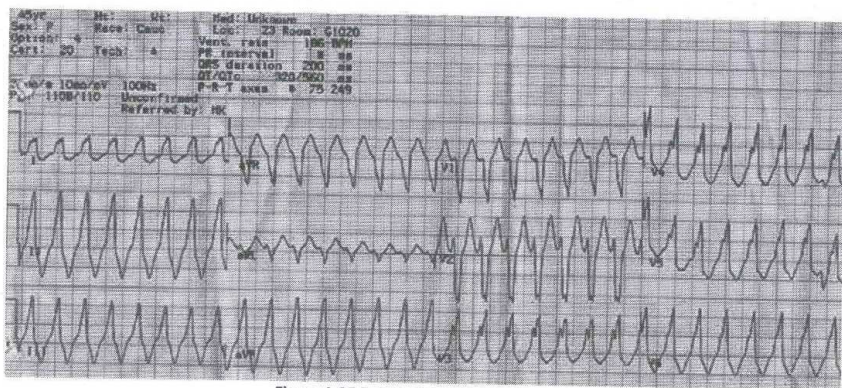


Figure 1.26.5. Broad complex tachycardia

- In a **45-year-old** woman with a structurally normal heart. Note the **left bundle**, right axis morphology of the QRS complex. The width of the QRS complex is **200 milliseconds**.
- The initial R wave in V1 is about 35 milliseconds in duration. There is a notch on the down stroke of the S wave in V1. All these morphologic features point to a **diagnosis of VT**.
- There is no precordial concordance in ECG leads V1 through V6 however, a feature more frequently seen in SVTs.
- Adenosine (12 mg IV) failed to terminate the tachycardia or change the rhythm.

2. NARROW-COMPLEX TACHYCARDIA

2.1. IRREGULAR NARROW-COMPLEX TACHYCARDIA

- An irregular narrow-complex tachycardia is most likely to be **AF** with an uncontrolled ventricular response or, less commonly, **atrial flutter with variable AV block**.
- The three main causes are:
 - *Atrial Fibrillation*
 - *Atrial flutter with variable block*
 - *Multifocal Atrial Tachycardia*

	BROAD COMPLEX TACHYCARDIA	NARROW COMPLEX TACHYCARDIA
IRREGULAR	<ul style="list-style-type: none"> ◦ AF with a BBB ◦ Pre-excited AF ◦ Torsades de pointes 	<ul style="list-style-type: none"> ◦ Atrial Fibrillation ◦ Atrial flutter with variable block ◦ Multifocal Atrial Tachycardia
DC SHOCK	Defibrillation dose, Not synchronised	120-200j biphasic or 200j monophasic
REGULAR	<ul style="list-style-type: none"> ◦ VT ◦ SVT with BBB ◦ Sinus tachycardia with BBB ◦ Atrial flutter with BBB 	<ul style="list-style-type: none"> ◦ Sinus tachycardia ◦ Atrial Flutter ◦ Re-entrant SVT
DC SHOCK	100j	50-100j

- **Atrial fibrillation:** there is no evidence of any organised atrial activity. Beware labelling coarse AF as flutter – the clue is the ‘flutter’ rate not being sufficiently fast. True flutter is demonstrated by atrial activity every 200 msec (i.e. every large square).
- **Atrial flutter with variable block:** Look hard for regular flutter waves. Note flutter with variable block is much rarer than AF (and not necessarily treated differently)
- **Multifocal atrial tachycardia:** Look for varying and irregular atrial activity – P waves of 3 different morphologies are needed to make the diagnosis. It is typically seen in **patients with decompensated lung disease**. The treatment is geared towards resolving the respiratory embarrassment rather than the tachycardia itself.
- Once atrial fibrillation is identified as the underlying arrhythmia, it should be treated as such the treatment of atrial fibrillation is discussed in detail in the relevant module.

ENERGY SELECTION FOR DEFIBRILLATION OR CARディオVERSION

- In 2010, the American Heart Association issued guidelines for initial energy requirements for monophasic and biphasic waveforms.
- **Atrial fibrillation:**
 - 200 Joules for monophasic devices
 - 120-200 Joules for biphasic devices
- **Atrial flutter:**
 - 100 Joules for monophasic devices
 - 50-100 Joules for biphasic devices
- **Ventricular tachycardia with pulse:**
 - 200 Joules for monophasic devices
 - 100 Joules for biphasic devices
- **Ventricular fibrillation or pulseless ventricular tachycardia:**
 - 360 Joules for monomorphic devices/ 120-200 Joules for biphasic devices

2.2. REGULAR NARROW-COMPLEX TACHYCARDIA

- o Sinus tachycardia
- o AVNRT and AVRT (paroxysmal supraventricular tachycardia)
- o Atrial flutter with regular AV conduction (often 2:1)

A. SINUS TACHYCARDIA

- Sinus tachycardia is not an arrhythmia.
- This is a common physiological response to stimuli such as exercise or anxiety.
- In a sick patient, it may occur in response to many conditions including pain, infection, anaemia, blood loss, and heart failure.
- Treatment is directed at the underlying cause. Trying to slow sinus tachycardia that has occurred in response to most of these conditions will usually make the situation worse.
- *Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.*

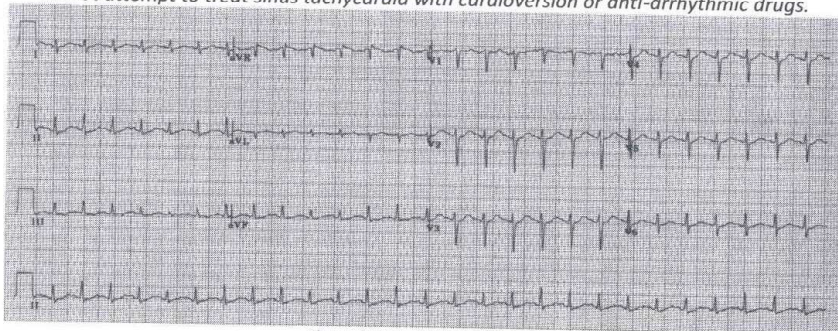


Figure 1.26.6. Sinus Tachycardia

B. PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

- **AV nodal re-entry tachycardia** is the commonest type of paroxysmal supraventricular tachycardia (SVT), often seen in people without any other form of heart disease.
- It is rare in the peri-arrest setting. It causes a regular, narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. The heart rate is commonly well above the typical range of sinus rhythm at rest (60–100/min).
- It is usually benign (unless there is additional, co-incidental, structural heart disease or coronary disease) but it may cause symptoms that the patient finds frightening.
- AV re-entry tachycardia occurs in patients with the **WPW syndrome**, and is also usually benign, unless there is additional structural heart disease.
- The common type of AVRT is a **regular narrow-complex tachycardia**, usually having no visible atrial activity on the ECG.

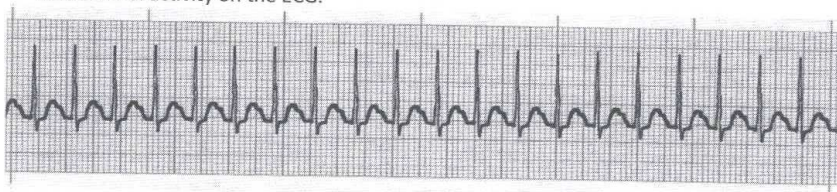


Figure 1.26.7. Supraventricular Tachycardia

C. ATRIAL FLUTTER WITH REGULAR AV CONDUCTION (often 2:1)

- This produces a regular narrow-complex tachycardia.
- It may be difficult to see atrial activity and identify flutter waves in the ECG with confidence, so the rhythm may be indistinguishable, at least initially, from AVNRT or AVRT.
- Typical atrial flutter has an atrial rate of about **300/min**, so atrial flutter with 2:1 conduction produces a tachycardia of about **150/min**.
- Much faster rates (>160/min) are unlikely to be caused by atrial flutter with 2:1 conduction.
- Regular tachycardia with slower rates (e.g. 125–150/min) may be due to atrial flutter with 2:1 conduction, usually when the rate of the atrial flutter has been slowed by drug therapy.

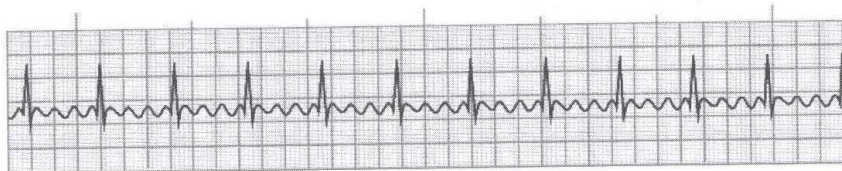


Figure 1.26.8. Atrial Flutter

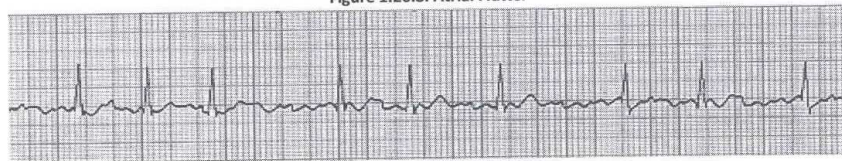


Figure 1.26.9. Atrial fibrillation

• TREATMENT OF REGULAR NARROW-COMPLEX TACHYCARDIA

- o If the patient is **unstable (compromised)**: **Synchronised DC cardioversion**.
- o It is reasonable to apply **vagal manoeuvres** and/or give **adenosine** to an unstable patient with a regular narrow-complex tachycardia while preparations are being made urgently for synchronised cardioversion. Do not delay electrical cardioversion if adenosine fails to restore sinus rhythm.
- o **In the absence of adverse features (Not compromised)**:
 - Start with **vagal manoeuvres**.
 - If the arrhythmia persists and is not atrial flutter, give **Adenosine 6 mg as a rapid IV bolus**.
 - If there is no response (i.e. no transient slowing or termination of the tachyarrhythmia) to adenosine 6 mg IV, **give a 12 mg IV bolus**.
 - If there is no response give one further **12 mg IV bolus**.
 - If adenosine is contra-indicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, consider giving **Verapamil 2.5–5 mg IV over 2 min**.
- Vagal manoeuvres or adenosine will terminate almost all AVNRT or AVRT within seconds. Failure to terminate a regular narrow-complex tachycardia with adenosine suggests an atrial tachycardia such as **atrial flutter** (unless the adenosine has been injected too slowly or into a small peripheral vein).

- **Other Drugs in SVT:**
 - Amiodarone
 - Beta blockers
 - Sotalol
 - Flecainide
 - Digoxin – not in uni or multifocal atrial tachycardia or AV dependent arrhythmias
 - Verapamil – **not in AV node re-entry tachycardia**

ADENOSINE CONTRAINDICATIONS:

- Hypersensitivity
- 2nd or 3rd degree AV block (except those on pacemakers),
- Sick sinus syndrome,
- Atrial fibrillation,
- V-tach
- Bronchoconstrictive or bronchospastic lung disease (eg, asthma)

3. VENTRICULAR FIBRILLATION

INTRODUCTION

- Ventricular fibrillation (VF) is the the most important shockable cardiac arrest rhythm.
- The ventricles suddenly attempt to contract at rates of up to **500 bpm**.
- This rapid and irregular electrical activity renders the ventricles unable to contract in a synchronised manner, resulting in immediate loss of cardiac output.
- The heart is no longer an effective pump and is reduced to a quivering mess.
- Unless advanced life support is rapidly instituted, this rhythm is invariably fatal.
- Prolonged ventricular fibrillation results in decreasing waveform amplitude, from initial coarse VF to fine VF and ultimately **degenerating into asystole** due to progressive depletion of myocardial energy stores.

CAUSES

- Myocardial ischemia / infarction
- Electrolyte abnormalities
- Cardiomyopathy (dilated, hypertrophic, restrictive)
- Long QT (acquired / congenital) causing TdP → VF
- Brugada syndrome
- Drugs (e.g. verapamil in patients with AF+WPW)
- Environmental – electrical shocks, drowning, hypothermia
- Pulmonary embolism
- Cardiac tamponade
- Blunt trauma (Comotio Cordis)

ECG FINDINGS

- Chaotic irregular deflections of varying amplitude
- No identifiable P waves, QRS complexes, or T waves
- Rate 150 to 500 per minute
- Amplitude decreases with duration (coarse VF → fine VF)
- ***VF should never be diagnosed from the 12-lead ECG!***

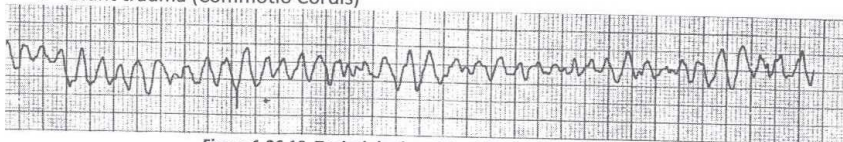


Figure 1.26.10. Typical rhythm strip of Ventricular Fibrillation

II. BRADYCARDIA**Introduction**

- Bradycardia is defined as a heart rate of less than **60 beats per minute**.

CAUSES OF BRADYCARDIA

Physiological	<ul style="list-style-type: none"> ○ Athletes
Intrinsic cardiac causes	<ul style="list-style-type: none"> ○ Idiopathic degeneration ○ Infarction and Ischemia ○ Myotonic dystrophy ○ Congenital Heart disease ○ Sick sinus syndrome
Infections	<ul style="list-style-type: none"> ○ Endocarditis
Autonomic mediated	<ul style="list-style-type: none"> ○ Vasovagal syncope ○ Carotid sinus hypersensitivity ○ Cough or Micturition syncope
Systemic Illness	<ul style="list-style-type: none"> ○ Hyperthyroidism ○ Hypothermia ○ Sarcoidosis ○ Amyloidosis
Head Injury	<ul style="list-style-type: none"> ○ Cushing's syndrome
Electrolytes disturbance	<ul style="list-style-type: none"> ○ Hyper/hypokalaemia
Drugs	<ul style="list-style-type: none"> ○ Betablockers and CCB ○ Digoxin ○ Organophosphates ○ Clonidine ○ Quinidine, Amiodarone

- Bradycardia may present as an incidental finding or with symptoms related to hypotension.
- Other symptoms may relate to the underlying cause:
 - Pre-syncope
 - Syncope
 - Nausea
 - Breathlessness
 - Weakness or fatigue

THE DIFFERENT TYPES OF BRADYCARDIA

- Sinus bradycardia
- Sinus arrest
- Junctional Bradycardia
- Atrioventricular Blocks:
 - First degree
 - Second degree (Mobitz type I and II)
 - Third degree (Complete AV Block)

1. ATRIOVENTRICULAR BLOCK

- AV block occurs when atrial depolarisation fails to reach the ventricles because of a block involving the AV node or the His-Purkinje system.
- If block is at the AV nodal level complexes will be narrow.
- If block is lower down in the His-Purkinje system complexes will be wide.
- The higher the block the more likely it will respond to increases in sympathetic tone or the use of atropine.
- Three degrees of block are recognised and described below:

1. FIRST DEGREE AV BLOCK

- PR interval > 0.20 sec; all P waves conduct to the ventricles.

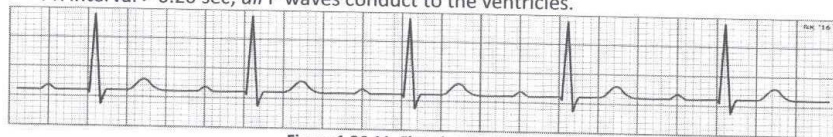


Figure 1.26.11. First degree AV Block

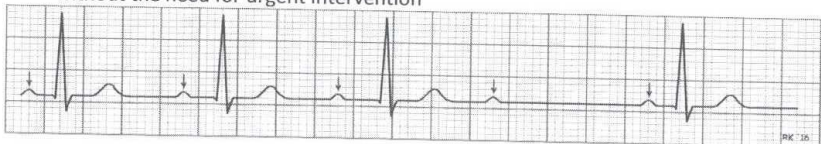
- This may be a sign of **early fibrosis or ischaemia** in the AV node but is most commonly a normal variant and is asymptomatic.
- In the context of an acute coronary syndrome it requires monitoring in case of progression to other forms of heart block.
- *It does not require treatment.*

2. SECOND DEGREE AV BLOCK

- The QRS remains narrow but atrial impulses fail to conduct normally to the ventricles in one of the following ways:

A. MOBITZ TYPE I (WENCKEBACH)

- The PR interval lengthens progressively after each successive P wave until a P wave is not conducted.
- This is common following **inferior acute myocardial infarction (AMI)** when it may progress to complete heart block.
- Mobitz type 1 heart block (Wenckebach) is normally asymptomatic and resolves without the need for urgent intervention



B. MOBITZ TYPE II

- There is a constant PR interval but some P waves fail to conduct to the ventricles
- The ratio of conducted and non-conducted beats may be fixed (e.g. 2:1 or 3:1).
- This is less common than Mobitz type I, often symptomatic and of more concern.
- It signifies **septal involvement** in the setting of **AMI** and commonly progresses to complete heart block.
- Patients who have this diagnosed on pre-operative assessment are fitted with pacemakers before undergoing anaesthesia.

- **Mobitz type 2 heart block commonly progresses to complete heart block which may require urgent intervention.**
- In Type II block several consecutive P waves may be blocked as illustrated below:

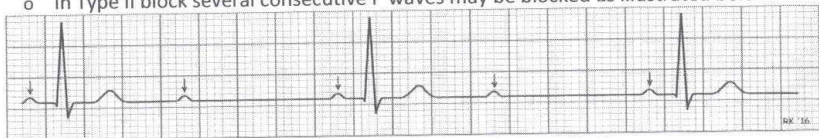
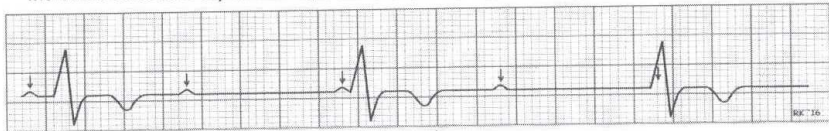


Figure 1.26.12. Second degree heart blocks Mobitz II

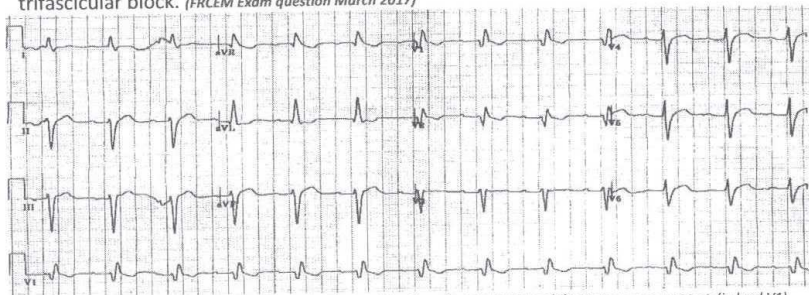
3. THIRD DEGREE AV BLOCK (COMPLETE HEART BLOCK)

- All P waves fail to conduct to the ventricles resulting in a broad complex ventricular escape rhythm.
- A rhythm originating in the high septal region will have a rate of 40-50 beats per minute. If originating from a lower ventricular site, the rate will be lower at 30-40 beats per minute.
- Although this may be a coincidental finding it usually presents with lethargy and syncope.
- It signifies **significant fibrosis or ischaemia** in the AV node and **requires a permanent pacemaker**. Following an **anterior AMI**, it indicates extensive damage to the septal region and indicates a worse prognosis. *Complete heart block in the setting of acute anterior MI indicates extensive septal damage and is a poor prognostic sign.*



4. TRIFASCICULAR BLOCK

- A trifascicular block is the combination of:
 - **A right bundle branch block,**
 - **Left anterior or posterior fascicular block and**
 - **A first-degree AV block (prolonged PR interval).**
- A trifascicular block is a precursor to complete heart block. Trifascicular block is usually present in various heart diseases (it is definitely not a normal finding) and sometimes can progress into the **third-degree AV block**. While a trifascicular block itself does not require any treatment, high doses of AV blocking agents likely should be avoided.
- Some series report a 50% lifetime need for a **permanent pacemaker** in the setting of a trifascicular block. (FRCM Exam question March 2017)



This ECG record of a trifascicular block contains the combination of LAFB, RBBB and the first-degree AV block (in lead V1).

ED MANAGEMENT OF BRADYCARDIAS

- The ALS bradycardia algorithm recommends that patients are initially assessed using the ABCDE approach and the following are carried out:
 - *Monitor SpO₂ and give oxygen if hypoxic*
 - *Monitor ECG and BP, and record 12-lead ECG*
 - *Obtain IV access*
 - *Identify and treat reversible causes (e.g. electrolyte abnormalities)*
- The four 'adverse features' listed by the ALS bradycardia algorithm are:
 - *Shock/ Syncope/ Myocardial infarction/ Heart failure*
- If any of these adverse features are present, then 500 mcg IV atropine should be administered.
- If there is not a satisfactory response, then interim measures should be considered:
 - *Repeated atropine doses up to a maximum of 3 mg or;*
 - *Transcutaneous pacing or;*
 - *Isoprenaline, adrenaline or alternative drugs*

2010 Resuscitation Guidelines

Resuscitation Council (UK)

Adult bradycardia algorithm

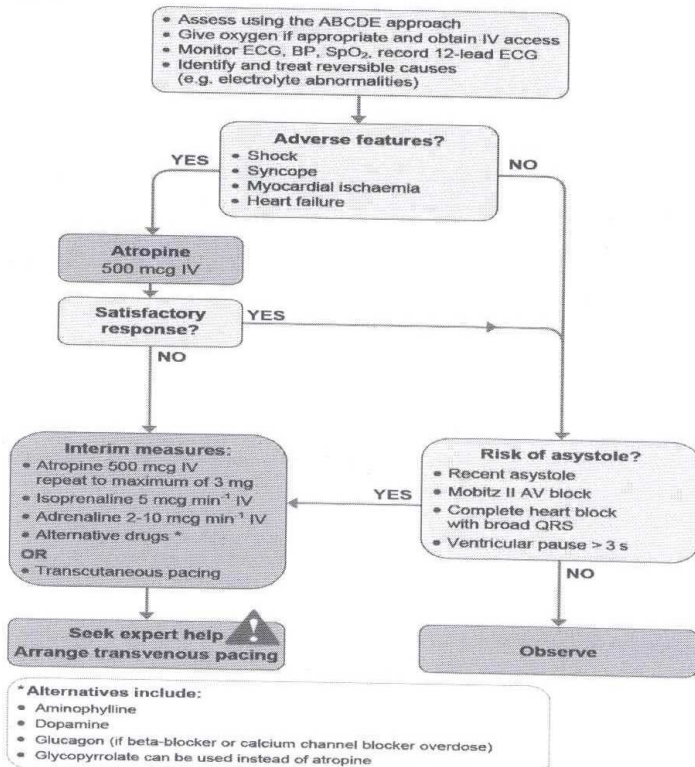


Figure 1.26.13. Bradycardia Algorithm

III. ATRIAL FIBRILLATION

• DEFINITION

- o Atrial Fibrillation (AF) is an atrial tachydysrhythmia characterised by predominantly uncoordinated atrial activation with consequent deterioration of atrial mechanical function.
- o *The p waves which represent depolarisation of the atria, are absent during atrial fibrillation and the heart rhythm is irregularly irregular.*
- o The main goals of treatment of atrial fibrillation are to minimize circulatory instability or insufficiency and to prevent stroke.
- o Circulatory instability or insufficiency are normally managed by either a rate or rhythm control strategy.
- o In an emergency, when circulatory collapse is imminent due to an unacceptably high ventricular rate **immediate cardioversion** may be indicated.
- o The risk of stroke must be assessed in all AF patients and treated accordingly.

CLINICAL ASSESSMENT

• Assessment of time of rhythm onset

- o The most important ED determination in AF is the probable **duration of the dysrhythmia**.
- o The currently recognised classification of AF relates to the duration and persistence of the AF:
 - **Paroxysmal** = episode of AF that terminates spontaneously
 - **Persistent** = episode of AF that requires cardioversion
 - **Permanent** = AF is resistant to multiple cardioversions
- o Many of the patients who present to the Emergency Department in AF have had the condition diagnosed previously and been commenced on appropriate treatment, **ie rate or rhythm control**.
- o However, some will not previously been known to be in AF and will therefore require a full assessment with a view to commencing therapy.
- o Some patients present with what has often been called **"Fast AF."** This is a misnomer since all patients in AF have chaotic atrial electrical activity with no discernible pattern, so the description "fast" which implies a contradistinction to "slow" is incorrect.
- o The correct description is **AF with a fast /slow / controlled ventricular response**.

• Assessment of precipitating events

- o There are many illnesses which may precipitate new atrial fibrillation or worsen the cardiovascular consequences of pre-existing AF.
- o Alcohol is also a common precipitant – the so called **"holiday heart."**

CARDIAC PRECIPITANTS	NON-CARDIAC PRECIPITANTS
<ul style="list-style-type: none"> o Ischaemic heart disease o Heart failure o Hypertension o Valvular heart disease (commonly mitral) o Sick sinus syndrome o Pericarditis o Cardiomyopathy 	<ul style="list-style-type: none"> o Hyperthyroidism o Pulmonary embolus o Sepsis o Alcohol excess or withdrawal o Hypokalaemia o Hypothermia o Drug use (cocaine)

NICE GUIDANCE ON STROKE RISK STRATIFICATION

CHA2DS2 VASC SCORE

THROMBOEMBOLIC/STROKE RISK

	Condition	Points
C	Congestive heart failure (or Left ventricular systolic dysfunction)	1
H	Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	1
A ₂	Age ≥ 75 years	2
D	Diabetes Mellitus	1
S ₂	Prior Stroke or TIA or thromboembolism	2
V	Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)	1
A	Age 65–74 years	1
Sc	Sex category (i.e. female sex)	1

- **A score of 0 in men or 1 in female:** low risk and no anticoagulation is required.
- **A score of 1 in men only:** moderate risk, anticoagulant should be considered.
- **If the score is 2 or greater (male and female):** the patient is high risk, and the patient should be anticoagulated if there are no contraindications.
- Anticoagulation may be with **Apixaban, Dabigatran Etxilate, Rivaroxaban** or **Warfarin**
- **Do not offer Aspirin** monotherapy solely for stroke prevention to people with atrial fibrillation.

HAS BLED SCORE

RISK OF BLEEDING

	Condition	Points
H	Hypertension: (uncontrolled, >160 mmHg systolic)	1
A	Abnormal renal function: Dialysis, transplant, Cr >2.26 mg/dL or >200 μ mol/L	1
	Abnormal liver function: Cirrhosis or Bilirubin >2 x Normal or AST/ALT/AP >3 x Normal	1
S	Stroke: Prior history of stroke	1
B	Bleeding: Prior Major Bleeding or Predisposition to Bleeding	1
L	Labile INR: (Unstable/high INRs), Time in Therapeutic Range $< 60\%$	1
E	Elderly: Age > 65 years	1
D	Prior Alcohol or Drug Usage History (≥ 8 drinks/week)	1
	Medication Usage Predisposing to Bleeding: (Antiplatelet agents, NSAIDs)	1

- **A score of 3 or more:** indicates an increased risk of bleeding when anticoagulated that warrants caution or more regular review of the patient.
- **INVESTIGATION**
 - Full blood count, coagulation, U&E, LFT, TFT, Inflammatory markers
 - Chest X ray, ECG
 - ECHO to document LA diameter, LV systolic function, any evidence of valvular abnormality, or cardiac pathology

NICE CG180 AFIB RECOMMENDATIONS

- For a patient with AF, it is desirable to restore sinus rhythm within the 48-hour time period (from onset). In this instance, **no further anticoagulation or further in-hospital intervention is required.**
- However, where the AF has continued for longer than 48-hours, restoration of sinus rhythm risks **dislodging thrombi from the left atrial appendage.** In this instance, treatment is limited to **determining stroke risks and controlling the ventricular rate.**
- **RATE AND RHYTHM CONTROL**
 - **Sign of life-threatening haemodynamic instability:** DC Cardioversion stat
 - **No sign of life-threatening haemodynamic instability:**
 - **Less than 48hrs:** offer rate or rhythm control
 - **More than 48 hours or is uncertain:** start rate control
 - Consider either pharmacological or electrical cardioversion depending on clinical circumstances and resources in people with new-onset atrial fibrillation who will be treated with a rhythm control strategy.
 - If pharmacological cardioversion has been agreed on clinical and resource grounds for new-onset atrial fibrillation, offer:
 - A choice of **Flecainide or Amiodarone** to people with no evidence of structural or ischaemic heart disease or
 - **Amiodarone** to people with evidence of structural heart disease.
 - In people with atrial fibrillation in whom the duration of the arrhythmia is greater than 48 hours or uncertain and considered for long-term rhythm control, **delay cardioversion until they have been maintained on therapeutic anticoagulation for a minimum of 3 weeks.** During this period offer rate control as appropriate.
 - **Do not offer magnesium or a calcium-channel blocker** for pharmacological cardioversion.

WHEN TO OFFER RATE OR RHYTHM CONTROL

- **Offer rate control** as the first-line strategy to people with atrial fibrillation, except in people:
 - *Whose atrial fibrillation has a reversible cause*
 - *Who have heart failure thought to be primarily caused by atrial fibrillation*
 - *With new-onset atrial fibrillation*
 - *With atrial flutter whose condition is considered suitable for an ablation strategy to restore sinus rhythm*
 - *For whom a rhythm control strategy would be more suitable based on clinical judgement.*

RATE CONTROL

- Offer either a standard beta-blocker (that is, a beta-blocker other than sotalol) or a rate-limiting calcium-channel blocker as initial monotherapy to people with atrial fibrillation who need drug treatment as part of a rate control strategy. Base the choice of drug on the person's symptoms, heart rate, comorbidities and preferences when considering drug treatment. Consider digoxin monotherapy for people with non-paroxysmal atrial fibrillation **only if they are sedentary** (do no or very little physical exercise).
- If monotherapy does not control symptoms, and if continuing symptoms are thought to be due to poor ventricular rate control, consider combination therapy with any 2 of the following: **A beta-blocker, Diltiazem and Digoxin.**

- | IV Route | | PO Route | |
|------------|------------|------------|-----------------------------------|
| Metoprolol | 2.5-5mg IV | Bisoprolol | 2.5-10mg PO, Atenolol 25-100mg PO |
| Verapamil | 5mg IV | Diltiazem | 60-360mg PO tds |
| Digoxin | 0.5-1mg IV | Digoxin | 0.125-0.5mg PO |



- Consider **pharmacological and/or electrical rhythm control** for people with atrial fibrillation whose symptoms continue after heart rate has been controlled or for whom a rate-control strategy has not been successful.
- If pharmacological cardioversion has been agreed on clinical and resource grounds for new-onset atrial fibrillation, offer:
 - A choice of **Flecainide** or **Amiodarone** to people **with no evidence of structural or ischaemic heart disease**
 - **Amiodarone** to people with evidence of structural heart disease

WHEN TO OFFER EMERGENCY CARDIOVERSION

- Carry out emergency electrical cardioversion, without delaying to achieve anticoagulation, in people with **life-threatening haemodynamic instability caused by new-onset atrial fibrillation**.

CARDIOVERSION

- For people having cardioversion for atrial fibrillation that has persisted for longer than 48 hours:
 - Offer electrical (rather than pharmacological) cardioversion.
 - Consider **amiodarone therapy starting 4 weeks before** and continuing for **up to 12 months** after electrical cardioversion to maintain sinus rhythm, and discuss the benefits and risks of amiodarone with the person.
- For people with atrial fibrillation of greater than 48 hours' duration, in whom elective cardioversion is indicated:
 - Both **transoesophageal echocardiography-guided cardioversion and conventional cardioversion** should be considered equally effective
 - A transoesophageal echocardiography-guided cardioversion strategy should be considered:
 - Where experienced staff and appropriate facilities are available and
 - Where a minimal period of precardioversion anticoagulation is indicated due to the person's choice or bleeding risks.

ANTICOAGULATION

- **Do not offer aspirin monotherapy** solely for stroke prevention to people with atrial fibrillation.
 - In people with **new-onset atrial fibrillation** who are receiving no, or subtherapeutic, anticoagulation therapy:
 - In the absence of contraindications, **offer heparin at initial presentation**
 - Continue heparin until a full assessment has been made and appropriate antithrombotic therapy has been started, based on risk stratification.
 - In people with a **confirmed diagnosis of atrial fibrillation of recent onset** (less than 48 hours since onset), offer oral anticoagulation if:
 - Stable sinus rhythm is not successfully restored within the same 48-hour period following onset of atrial fibrillation or
 - There are factors indicating a high risk of atrial fibrillation recurrence
 - In people with new-onset atrial fibrillation where there is uncertainty over the precise time since onset, **offer oral anticoagulation as for persistent atrial fibrillation**.

❖ **Consider amiodarone for people with left ventricular impairment or heart failure**

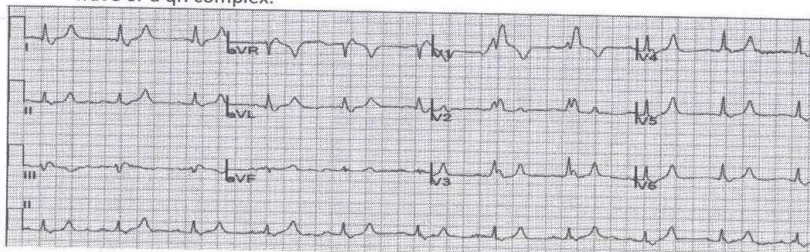
❖ **Do not offer class 1c antiarrhythmic drugs such as flecainide or propafenone to people with known ischaemic or structural heart disease.**

IV. INTRAVENTRICULAR BLOCKS

A. RIGHT BUNDLE BRANCH BLOCK (RBBB)

1. "COMPLETE" RBBB

- o **Diagnostic Criteria**
 - Broad QRS > 120 ms
 - RSR' pattern in V1-3 ('M-shaped' QRS complex)
 - Wide, slurred S wave in the lateral leads (I, aVL, V5-6)
- o **Associated Features**
 - ST depression and T wave inversion in the right precordial leads (V1-3)
- o **Variations**
 - Sometimes rather than an RSR' pattern in V1, there may be a broad monophasic R wave or a qR complex.



- The frontal plane QRS axis in RBBB should be in the normal range (i.e., -30 to +90 degrees).
 - o If left axis deviation is present, think about **left anterior fascicular block**
 - o If right axis deviation is present, think about **left posterior fascicular block** in addition to the RBBB.

2. "INCOMPLETE" RBBB

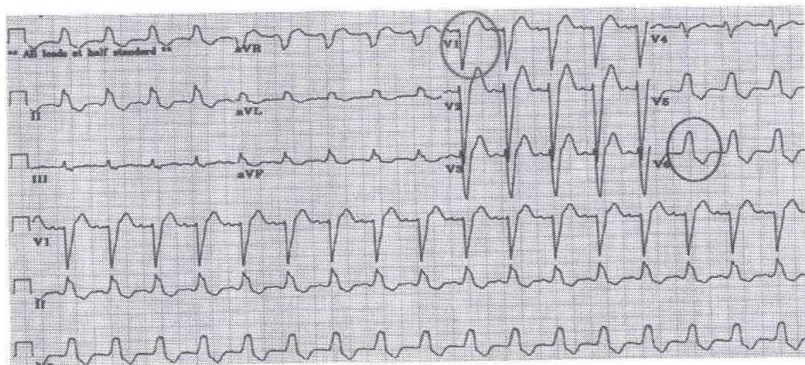
- o QRS duration of **0.10 - 0.12s** with the same terminal QRS features.
- o This is often a normal variant.
- o The "normal" ST-T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards.
- o If the ST-T waves are in the *same direction* as the terminal QRS forces, they should be labeled **primary ST-T wave abnormalities**

ECG DIAGNOSIS OF BUNDLE BRANCH BLOCK

- o QRS > 0.12 sec
- o Look at V1:
 - **Terminal R** = RBBB as excitation spreading from left to right
 - **Terminal S** = LBBB as excitation spreading away from right
- o Confirm I: (& aVL V5&6)
 - **Terminal S** = RBBB as excitation going away from left side
 - **Terminal R** = LBBB as excitation heading towards left
 - The above equates to pattern recognition of **MaRow/ William** in V1-6.
 - With LBBB associated ST/T opposite to QRS, poor R progression in V1-6, RS in V5, 6 left axis deviation.

B. LEFT BUNDLE BRANCH BLOCK (LBBB)**1. "COMPLETE" LBBB"**

- o **Diagnostic Criteria**
 - QRS duration of > 120 ms
 - Dominant S wave in V1
 - Broad monophasic R wave in lateral leads (I, aVL, V5-V6)
 - Absence of Q waves in lateral leads (I, V5-V6; small Q waves are still allowed in aVL)
 - Prolonged R wave peak time > 60 ms in left precordial leads (V5-6)
- o **Associated Features**
 - Appropriate discordance: the ST segments and T waves always go in the opposite direction to the main vector of the QRS complex
 - Poor R wave progression in the chest leads
 - Left axis deviation

**2. "INCOMPLETE" LBBB:**

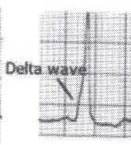
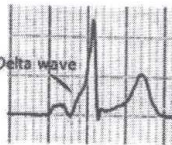
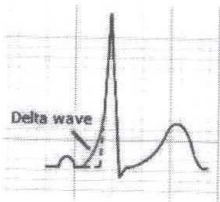
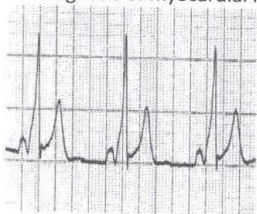
- o Looks like LBBB but QRS duration = **0.10 to 0.12s**, with less ST-T change. This is often a progression of LVH.
 - Increased QRS voltage in the limb leads
- **Diagnosing AMI in LBBB**
 - o The Sgarbossa criteria only apply in LBBB (see rules below)
 - o In true LBBB, there must not be any Q wave in the lateral leads

SGARBOSSA CRITERIA

- **Of acute MI with LBBB (any of following)**
 - o ST elevation ≥ 1 mm concordant with QRS
 - o ST depression ≥ 1 mm in V1-3
 - o ST elevation ≥ 5 mm discordant with QRS

C. WOLFF-PARKINSON-WHITE PREEXCITATION

- o QRS complex represents a **fusion** between two ventricular activation fronts:
 - Early ventricular activation in region of the accessory AV pathway (**Bundle of Kent**)
 - Ventricular activation through the normal AV junction, bundle branch system
- o **ECG criteria include all of the following:**
 - Short PR interval ($< 0.12s$)
 - Initial slurring of QRS complex (**delta wave**) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway
 - Prolonged QRS duration (usually $> 0.10s$)
 - Secondary ST-T changes due to the altered ventricular activation sequence
- o QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).
- o **Delta waves**, if negative in polarity, may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.



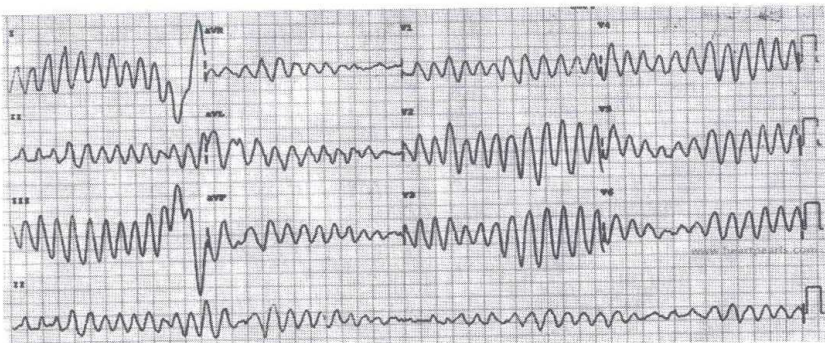
D. ATRIAL FIBRILLATION & ATRIAL FLUTTER IN WPW

- Atrial fibrillation can occur in up to 20% of patients with WPW.
- Atrial flutter can occur in up to 7% of patients with WPW.
- The accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node.
- Rapid ventricular rates may result in degeneration to VT or VF.
- **ECG features of Atrial Fibrillation in WPW are:**
 - o Rate > 200 bpm
 - o Irregular rhythm
 - o Wide QRS complexes due to abnormal ventricular depolarisation via accessory pathway
 - o QRS Complexes change in shape and morphology
 - o Axis remains stable unlike **Polymorphic VT**
- *Atrial Flutter results in the same features as AF in WPW except the rhythm is regular and may be mistaken for VT.*
- **Treatment of AF with WPW**
 - o In a **haemodynamically unstable** patient urgent synchronised DC cardioversion is required.
 - o Medical treatment options in a **stable** patient include **Procainamide** or **Ibutilide**, although DC cardioversion may be preferred.
 - o Treatment with AV nodal blocking drugs e.g. **adenosine**, **calcium-channel blockers**, **beta-blockers** may increase conduction via the accessory pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF.

V. TORSADES DE POINTES

• BACKGROUND

- o Torsade de pointes (TdP) is a form of polymorphic ventricular pro-arrhythmia.
- o Associated with QT interval prolongation and prominent U waves on resting ECG
- o ECG = prolonged re-polarisation and so, early after depolarisation (EAD)
- o Can be congenital
- o Usually acquired due to **potassium channel dysfunction**.
- o It may degenerate to ventricular fibrillation.



• PHYSIOLOGY

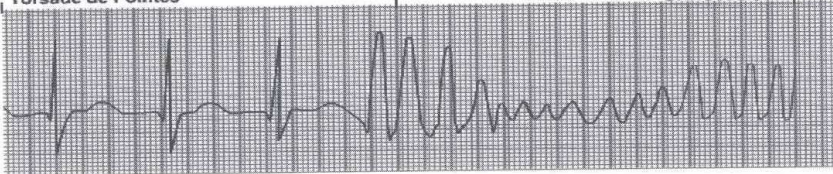
- o Ventricular re-polarisation is initiated by exodus of intracellular K⁺.
- o Drugs can block this K⁺ channel - delaying repolarisation (prolonging Q-T interval).
- o Other factors are
 - Female
 - ↑ Age
 - Electrolyte disturbance
 - CCF, Bradycardia, Ischaemia Congenital
 - Main drug culprits

• DRUG CAUSES

- o Antiarrhythmics especially Class Ia and III.
- o Phenothiazines and butyrophenones.
- o Tricyclic antidepressants.
- o Non-sedative antihistamines.
- o Some antibiotics especially macrolides and anti-fungals.
- o Organophosphates.
- o Cocaine
- o Electrolyte abnormalities (hypokalaemia, hypomagnesaemia)

Torsade de Pointes

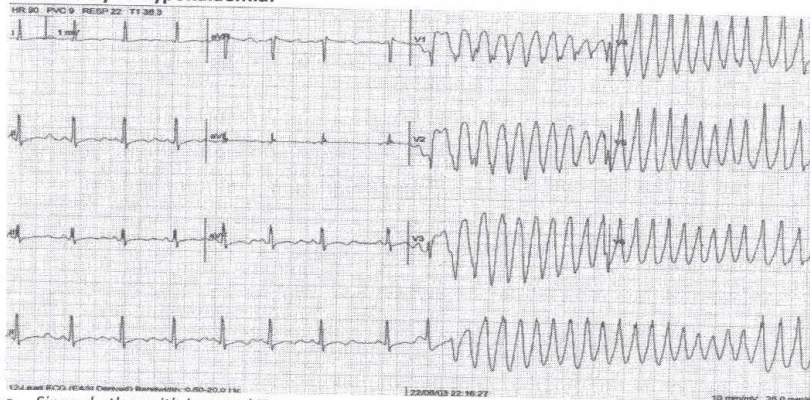
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TREATMENT

- To treat haemodynamic compromise immediately.
- To alter the after-depolarisation effect.
- To shorten the QT interval.
- Haemodynamic compromise: immediate DC cardioversion:** 150-200J
- Magnesium**, at a dose of **2g magnesium sulphate IV over 1-2min**, is used to suppress EAD's in the emergency situation. The serum magnesium level need not be known prior to treatment.
- Correction of hypokalaemia** to a serum K⁺ concentration of > 4.5 mmol/l also helps suppress EAD's.
- Lignocaine** has been used. However its effect is inconsistent with a reported success rate of only 50%.
- Cardiac pacing at 100-140/min is the treatment of choice.** The basic heart rate should be accelerated, as there is an inverse relationship between rate and the re-polarisation duration.
- Isoprenaline** should only be a temporising measure as it can promote EADs.
- Involve a cardiologist early.

TdP secondary to hypokalaemia:



- Sinus rhythm with inverted T waves, prominent U waves and a long Q-U interval due to severe hypokalaemia (K⁺ 1.7)*
- A premature atrial complex (beat #9 of the rhythm strip) lands on the end of the T wave, causing 'R on T' phenomenon and initiating a paroxysm of polymorphic VT.*
- Because of the preceding long QU interval, this can be diagnosed as TdP.*
- Polymorphic ventricular tachycardia (PVT)** is a form of ventricular tachycardia in which there are multiple ventricular foci with the resultant QRS complexes varying in amplitude, axis and duration. The commonest cause of PVT is **Myocardial Ischaemia**.
- Torsades de pointes (TdP)** is a specific form of polymorphic ventricular tachycardia occurring in the context of QT prolongation; it has a characteristic morphology in which the QRS complexes "twist" around the isoelectric line. For TdP to be diagnosed, the patient has to have **evidence of both PVT AND QT prolongation**.
- Bidirectional VT** is another type of polymorphic VT, most commonly associated with digoxin toxicity.

VI. WELLENS' SYNDROME

- Wellens' syndrome is a **preinfarction stage of coronary artery disease** and heralds an **impending extensive myocardial infarction of the anterior wall**.
- It is typified by **anginal chest pain**, **characteristic ECG changes** that usually occur after chest pain has resolved, and **negative cardiac biomarkers**.
- Wellens' syndrome presents as one of two characteristic T-wave abnormalities **seen in leads V2 and V3** on ECG:
 - Type A** (approximately 25% of cases) shows **biphasic T-waves**, with an initial positive deflection, and terminal negative deflection.
 - Type B** (approximately 75% of cases) shows **deeply inverted and symmetric T-waves**.
- The ST segment is seldom involved, but when it is, consists of ST elevation of less than 1 mm.

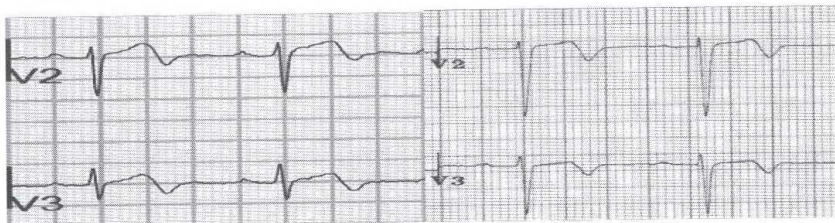


Figure 1.26.14. Type A wellen's syndrome: Biphasic T-waves in V2-V3

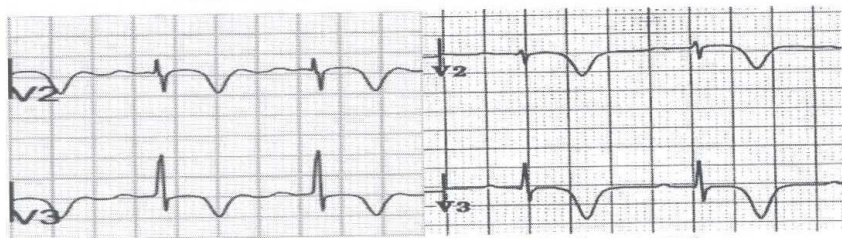


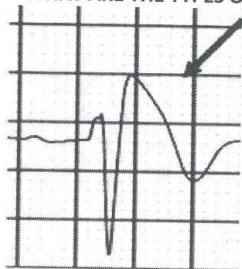
Figure 1.26.15. Type B well's syndrome: Deeply inverted T waves in V2-V3

- These changes are always **seen in leads V2 and V3**, but can be seen commonly in V4, less often in V1, and only occasionally seen in leads V5 and V6.
- Patients presenting with Wellens' syndrome will generally have signs and symptoms of typical anginal chest pain and **usually respond well to drug therapy** (nitrates and morphine).
- What is unusual is that the ECG changes that are typical of Wellens' syndrome typically appear after chest pain has resolved.*
- In fact, during an acute attack of chest pain, the T-wave abnormalities will normalize or become ST-segment elevation.*
- Left untreated, the patient presenting with Wellens' syndrome has a significant risk of **severe myocardial infarction and death**.
- In de Zwaan et al's initial study, of the patient's presenting with this ECG pattern who had myocardial infarction, the infarction occurred within 1 to 23 days (mean of 8.5 days) of admission.

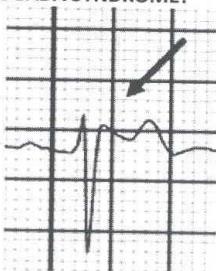
VII. BRUGADA SYNDROME

- **Brugada Syndrome** is an abnormal ECG (**Right Bundle Branch Block Pattern with coved ST elevation over the right precordial leads**), which leads to ventricular fibrillation (VF) and sudden cardiac death (SCD) in patients with structurally normal hearts.
- It has been recognized as a clinical entity since 1992.
- Why should all ED physicians know about this entity? Although a rare syndrome, it is often mistaken as a STEMI and more importantly the clinical spectrum can be asymptomatic to SCD.
- **WHO GETS BRUGADA SYNDROME?**
 - Males > Females in a 8 – 10: 1 ratio
 - Ages 20 – 40 years (There are case reports of age 2 days all the way up to 84 years)
 - Asian > US populations
 - Typically occurs at night, when there is a predominance of vagal activity.
- **HOW COMMON IS BRUGADA SYNDROME?**
 - Worldwide 4 – 12% of all sudden deaths
 - Type 1 Brugada occurs in 12/10,000 people
 - Type 2 and 3 Brugada occurs in 58/10,000 people
 - Prevalance of Brugada Pattern ECG: Asia (0.36%), Europe (0.25%), and in the USA (0.03%)
 - ECG pattern can wax and wane, making the true incidence underestimated.

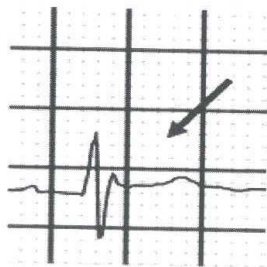
WHAT ARE THE TYPES OF BRUGADA SYNDROME?



Type 1



Type 2



Type 3

- **Sodium channel defect** that leads to impaired fast upstroke of phase 0 of the action potential.

AETIOLOGY

- ECG changes can be transient with Brugada syndrome and can also be unmasked or augmented by multiple factors:
 - Fever
 - Ischaemia
 - Hypokalaemia
 - Hypothermia
 - Post DC cardioversion
 - Multiple Drugs
 - Sodium channel blockers eg: Flecainide, Propafenone

- Calcium channel blockers
- Alpha agonists/ Beta Blockers/ Nitrates
- Cholinergic stimulation/ Cocaine/ Alcohol

• **ECG**

- o ECG changes may be intermittent and transient
- o Unusual or saddle-shaped ST elevation (>2mm) in leads V1 - V3
- o Partial or complete RBBB (+ T inversion)
- o J point elevation

• **Diagnostic Criteria**

- o Coved ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave is the only ECG abnormality that is **potentially** diagnostic. **This has been referred to as Brugada sign.**



Brugada sign

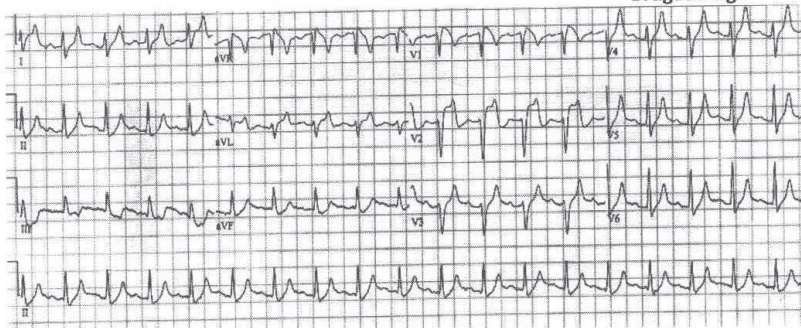


Figure 1.26.16. Brugada Type 1

• **WHERE IS THE MOST LIKELY ARRHYTHMOGENIC SUBSTRATE OF BRUGADA SYNDROME?**

- o Right Ventricular Outflow Tract (RVOT)
- o Only cardiac structure lying underneath 2nd and 3rd intercostal spaces
- o Brugada pattern may be absent in typical 4th intercostal space of leads V1 - V3.

• **WHAT IS THE BEST WAY TO RISK STRATIFY PATIENTS WITH BRUGADA SYNDROME?**

- o Symptomatic patients with recurrent syncope, agonal respirations at night during sleep, or unknown seizures are at the highest risk of dying.
- o Asymptomatic patients have an annual cardiac event rate of 0.25%, therefore there is little value in a risk stratification strategy to identify high risk patients

• **WHAT ARE THE TREATMENT OPTIONS FOR BRUGADA SYNDROME?**

- o **Quinidine** is the only medication that has shown benefit in prevention of VF and reduction of AICD shocks (Only 67% of patients can tolerate drug due to side effects)
- o **Implantable Cardiac Defibrillator (ICD):** Class 1 Indication in symptomatic patients (past history of VT/VF or syncope)
- o Defibrillator Versus B-Blocker in Unexplained Death in Thailand (DEBUT) Trial: Showed 0% death rate after ICD versus 18% in Beta Blocker group.
- o **Leadless ICDs:** 98% termination rate of VF/VT, but less pocket infection and lead revisions.
- o **Catheter Ablation:** Performed in 14 patients with no recurrent VF/VT with a median 32 month follow up.

VIII. LONG QT SYNDROME (LQTS)

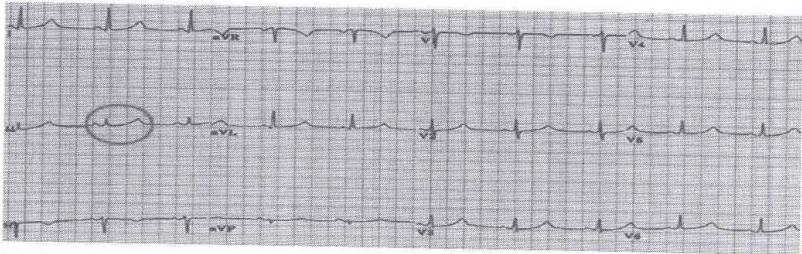
- Prolonged ventricular re-polarisation = prolongation of the QT interval
- Risk of Torsade de pointes and sudden death

Causes of a prolonged QTc (>440ms):

- **4H Must Raise Cardiac Pressure with Drugs**
 - Hypokalaemia
 - Hypomagnesaemia
 - Hypocalcaemia
 - Hypothermia
 - Myocardial ischemia
 - Raised intracranial pressure
 - Congenital long QT syndrome
 - Post-cardiac arrest
- **DRUGS: Triple AAA Tears First Endothelium**
 - Antihistaminics
 - Anticholinergics
 - Antiarrhythmics (specially Quinidine and Sotalol)
 - TCAS
 - Fluoroquinolones
 - Erythromycin
- **Other drugs are:** Chloroquine, Mefloquine, Haloperidol, Risperidone, Methadone, and HIV protease Inhibitors.
- **Congenital**
 - Romano-Ward syndrome - autosomal dominant.
 - Lange-Nielsen syndrome - autosomal recessive (assoc congenital deafness).
 - F > M, usually childhood or adolescence.
 - Once identified, first degree relatives should be screened.
- **Clinical Presentation**
 - Palpitations, syncope or near syncope, seizures, or cardiac arrest.

ECG findings

- $QTc = QT / \sqrt{R-R^2}$, >0.45 sec abnormal
- Abnormal T-wave (notched or biphasic)
- T-wave alternans



Treatment

- "Lifestyle modifications," (avoidance competitive sports and of all drugs known to prolong QT interval) (See above list).
- Treat with **B-blockers** (shorten the QT interval, reduce risk of Torsade and sudden death).
- High risk patients - **implantable cardioverter-defibrillators (ICDs)**
- Left cervicothoracic sympathectomy (block sympathetic to heart so reduce event rate).

CHAPTER 27. GYNAECOLOGY/OBSTETRICS IN ED**I. PELVIC PAIN****1. PELVIC INFLAMMATORY DISEASE****• BACKGROUND**

- Pelvic inflammatory disease (PID) is an infectious and inflammatory disorder of the upper female genital tract, including the uterus, fallopian tubes, and adjacent pelvic structures. Infection and inflammation may spread to the abdomen, including perihepatic structures (**Fitz-Hugh-Curtis syndrome**).
- The classic high-risk patient is a menstruating woman younger than 25 years who has multiple sex partners, does not use contraception, and lives in an area with a high prevalence of sexually transmitted disease (STD).
- PID is initiated by infection that ascends from the vagina and cervix into the upper genital tract.
- *Chlamydia trachomatis* is the predominant sexually transmitted organism associated with PID. Other organisms implicated in the pathogenesis of PID include *Neisseria gonorrhoeae*, *Gardnerella vaginalis*, *Haemophilus influenzae*, and anaerobes such as *Peptococcus* and *Bacteroides* species.
- Laparoscopic studies have shown that in 30-40% of cases, PID is polymicrobial.

• CAUSES OF PID

- **Sexually transmitted (90%):** Chlamydia, Gonorrhoea, Mycoplasma genitalium.
- **Non-sexually transmitted (10%, often post-surgical instrumentation):** *E. Coli*, Group B Strep, Bacteriodes, Gardenella.

• CLINICAL FEATURES OF PID

- Lower abdominal pain and tenderness.
- Abnormal vaginal or cervical discharge.
- Fever ($>38^{\circ}\text{C}$).
- Abnormal vaginal bleeding (intermenstrual, post-coital, or 'breakthrough').
- Deep dyspareunia.
- Cervical excitation.
- Adnexal tenderness \pm mass.

• INVESTIGATIONS IN PID

- Endocervical swabs for *Chlamydia* and *Gonorrhoea*.
- Urinary pregnancy test
- Bloods: ESR, CRP, and WCC are supportive but not specific.
- Transvaginal ultrasound may demonstrate inflamed/dilated Fallopian tubes or an abscess.

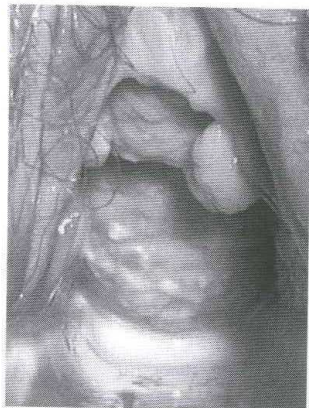
• ED MANAGEMENT OF PID

- **Outpatient:** CEFTRIAXONE 500MG IM/IV as single dose, then DOXYCYCLINE 100mg PO BD + METRONIDAZOLE 400mg PO TDS
- **Inpatient management** is indicated in the following circumstances:
 - Clinically severe disease
 - Tubo-ovarian abscess
 - Intolerance or lack of response to oral therapy
 - Surgical emergency not excluded.

- Inpatient antibiotic therapy is intravenous **Ceftriaxone 1g IV OD** and **DOXYCYCLINE 100mg PO BD + metronidazole 400mg PO TDS**.
- **Surgical drainage** may be required for tubo-ovarian abscesses.
- Consideration should be given to **removing an IUCD** in patients presenting with PID, especially if symptoms have not resolved within 72 hours.
- **Sexual partners** from the previous 6 months should be contacted and offered screening via the genitourinary medicine clinic.
- **COMPLICATIONS OF PID**
 - Infertility.
 - Ectopic pregnancy (five times increased risk).
 - Chronic pelvic pain.
 - Peritonitis.
 - Abscess formation.

2. VAGINAL CANDIDIASIS

- It is extremely common and approximately 90% of cases are caused by the fungus ***Candida albicans***.
- Risk factors for its development include:
 - Diabetes mellitus
 - Recent antibiotic treatment
 - Pregnancy
 - Immunosuppression
- Patients typically present with a **white 'cheesy' discharge, vaginal itching, dyspareunia and dysuria**.
- Examination will reveal **vulval erythema, oedema, satellite lesions** and sometimes associated **fissuring**.
- Treatment is with topical antifungals, such as **Clotrimazole and Miconazole**, is usually adequate.
- More severe cases sometimes require **Oral Fluconazole or Itraconazole**.



3. TRICHOMONAS VAGINALIS

- Infection is most commonly seen in sexually active females between the ages of 18 and 35 years.
- It is usually, but not always, acquired through sexual transmission.
- It typically presents with a profuse, offensive, **thin vaginal discharge**.
- The colour is usually **yellow or green**. It is often associated with vulval itching and soreness, dysuria, dyspareunia and abdominal pain.
- On examination, there will be **vulval and cervical erythema** and some patients will have a **'strawberry cervix'** where the ectocervix resembles the surface of a strawberry.
- The vaginal pH will be **> 4.5** in *Trichomonas vaginalis* infection.



- **Trichomonas vaginalis** infection is associated with:
 - Pelvic inflammatory disease
 - Increased risk of HIV infection
 - Preterm delivery and other pregnancy complications
- Treatment is with **Metronidazole** or **Tinidazole**.

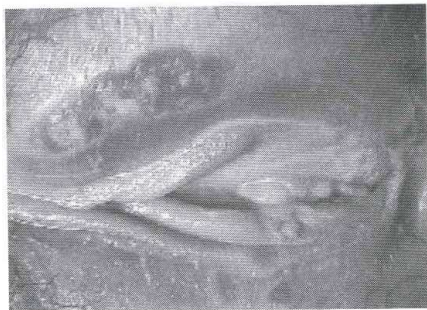
4. PRIMARY SYPHILIS

- Is caused by the spirochete bacterium *Treponema pallidum*.
- Primary syphilis is typically acquired via direct sexual contact with the infectious lesions of infected individual.
- The typical incubation period is 2-3 weeks but can be as long as 3 months.
- A primary lesion develops at the site of contact, initially as a **small painless nodule** that subsequently ulcerates and forms a **large painless ulcer**.
- The margins are typically indurated and red and there is often a clear serous discharge.
- Painless regional lymphadenopathy is also usually present.
- The treatment of choice for primary syphilis is long-acting procaine **Benzylpenicillin 600 mg daily by IMI for 10-12 days**.
- For CNS disease, secondary and tertiary syphilis, the treatment regime is for 14 days.



5. CHANCROID

- A sexually transmitted infection caused by the fastidious Gram-negative bacteria *Haemophilus ducreyi*.
- It is spread by direct sexual contact.
- Chancroid is relatively rare in the UK but is endemic in Africa, Asia and South America.
- HIV is an important co-factor, with a 60% association in Africa.
- The disease is characterized by the development of **painful ulcers on the genitalia**.
- In women the most common site of ulcer development is the labia majora.
- '**Kissing ulcers**' can develop where ulcers are situated in opposing surfaces of the labia.
- Painful lymphadenopathy occurs in 30-60% of patients and these can further develop into abscesses (buboes).



ED MANAGEMENT

- The CDC recommends a single oral dose of **1 gram of azithromycin** or a single IM dose of **ceftriaxone** for the treatment of chancroid.
- A **7 day course of oral Erythromycin** is an acceptable alternative.

- *Haemophilus ducreyi* is resistant to penicillins, tetracyclines, trimethoprim, ciprofloxacin, aminoglycosides and sulfonamides.
- **POTENTIAL COMPLICATIONS INCLUDE:**
 - Extensive adenitis
 - Large inguinal abscesses and/or sinuses
 - Phimosis
 - Superinfection with *Fusarium spp.* or *Bacteroides spp.*

6. BACTERIAL VAGINOSIS

- It is caused by an imbalance of naturally occurring bacterial flora within the vagina.
- Anaerobic organisms, such as *Gardnerella vaginalis*, *Mobiluncus spp.* and *Bacteroides spp.* proliferate and replace lactobacilli.
- *Gardnerella vaginalis* is the most commonly implicated bacteria.
- The commonest presenting symptom of BV is an **unpleasant, fishy-smelling discharge**.
- It is often worse after intercourse but there is not usually any accompanying vaginal soreness or irritation.
- Diagnosis can be made on the basis of **Amsel's criteria**, with any 3 of the following being required:
 - Thin, white or yellow, homogenous discharge
 - **Clue cells** (epithelial vaginal cells with a distinctive stippled appearance)
 - Vaginal pH > 4.5 (can be as high as 7.0)
 - **Positive 'whiff test'** (fishy odor released on addition of 10% potassium hydroxide to vaginal fluid)
- Treatment is with **Metronidazole** or **Clindamycin**.

7. VARICELLA ZOSTER AND PREGNANCY

- Varicella can cause serious complications in pregnant women.
- The risk of the foetus being affected is around 1% if the mother develops varicella in the first 28 weeks of pregnancy.
- The result is **foetal varicella syndrome (FVS)**, which is characterised by *eye defects, limb hypoplasia, skin scarring and neurological abnormalities*.
- Any pregnant woman who has not had chickenpox or who is found to be seronegative for **VZV IgG** should be advised to minimize any contact with chickenpox and shingles and to seek medical help immediately if exposed.
- If a pregnant woman is exposed, the first course of action is to perform a blood test and check for **VZV immunity**.
- If she is not immune and the history of the exposure is significant, she should be given **VZV immunoglobulin** as soon as possible. It is effective **up to 10 days after being exposed**.
- A pregnant woman that develops chickenpox should seek medical help urgently. There is an increased maternal risk of **Pneumonia, Encephalitis** and **Hepatitis** as well as the 1% risk of developing **FVS**.
- **Acyclovir** should be used with caution before 20 weeks gestation, but is recommended after 20 weeks if the woman presents within 24 hours of the onset of the rash.

II. MISCARRIAGE & ECTOPIC PREGNANCY

DEFINITIONS

- **Miscarriage** is the loss of a pregnancy before 23 completed weeks.
 - **Early miscarriage** is more precisely defined as pregnancy loss in the first 12 weeks.
 - **Late miscarriage** as pregnancy loss thereafter.
- **Ectopic pregnancy** occurs where a fertilized ovum is implanted in any tissue other than the uterine endometrium.
- **Antepartum haemorrhage (APH)** is defined as vaginal bleeding occurring from the 24th week of pregnancy and prior to the birth of the baby.
- **Postpartum hemorrhage (PPH)** is often defined as the loss of more than 500 ml or 1,000 ml of blood within the first 24 hours following childbirth.
- **Rhesus D antigen** is found on the surface of RBC and is capable of inducing intense antigenic reactions. Individuals without the antigen are determined rhesus negative and are homozygous recessive.

0-12 WEEKS	12-23 WEEKS	24 WEEKS- PREDELIVERY	24HRS TO 12 WEEKS POST DELIVERY
EARLY MISCARRIAGE	LATE MISCARRIAGE	APH	PPH

1. MISCARRIAGE

- Since 1997 the RCOG has encouraged the use of the term miscarriage rather than abortion.
- Miscarriage is subdivided as follows:
 - **Threatened miscarriage:** bleeding or cramping in a continuing pregnancy. The cervical os is closed. An ultrasound scan is required to confirm fetal heart activity.
 - **Complete miscarriage:** all the fetal material has passed and the uterus is empty. The cervical os will be closed and where there has not previously been an US scan, one should be performed together with serum hCG to confirm pregnancy failure.
 - **Incomplete miscarriage:** there is retained products of conception within the uterus and the os remains open. The patient is at risk of haemorrhage and infection.
 - **Early embryonic/fetal demise** (previously known as missed/anembryonic pregnancy/blighted ovum): a non-viable pregnancy at 12 weeks where the products of conception have not been passed.
 - **Miscarriage with infection** (previously referred to as septic): this is secondary to either a spontaneous miscarriage or induced termination. Presentation is with fever and foul-smelling discharge.
- **CAUSATIVE FACTORS**
 - Chromosomal abnormalities
 - Increasing maternal age
 - Smoking
 - Alcohol
 - Uterine abnormalities
 - Maternal infection
 - Co-morbidity
- **PRESENTATION**
 - **Vaginal bleeding:** ranging from occasional spotting to significant haemorrhage or cervical shock.
 - **Abdominal pain**

2. ECTOPIC PREGNANCY

OVERVIEW

- Ectopic pregnancy = fertilized ovum which implants outside the lining of the uterus
- **RISK FACTORS FOR ECTOPIC PREGNANCY**
 - History of previous IUCD
 - Maternal age of 35-44 years
 - Previous ectopic pregnancy
 - Previous pelvic or abdominal surgery
 - Pelvic Inflammatory Disease (PID)
 - Several induced abortions
 - Conceiving after having a tubal ligation or while an IUD is in place
 - Smoking
 - Endometriosis
 - Undergoing fertility treatments or using fertility medications
- **Special note: CORNUAL IMPLANTATION**
 - Patients with cornual implantation may rupture after 12 weeks with catastrophic blood loss.
 - These patients sometimes present with symptoms of **gastroenteritis**.
 - No single sign or combination of signs is diagnostic.
 - Half of identified ectopics are in women with no known risk factors.

Cornual implantation



CLINICAL FEATURES

- **History**
 - PV bleeding
 - Abdominal/Pelvic pain
 - 6-8 weeks LMP
 - Shoulder tip pain (large amount of bleeding)
 - Lightheadedness
 - Postural symptoms
- **Examination**
 - Adnexal tenderness and masses
 - State of cervix and material passing through it
 - Fetal heart (almost never heard in ectopic)

INVESTIGATIONS

- Beta-HCG (should almost double every 2 days)
- Bloods to rule out other causes of abdominal pain, Rh status
- MSU
- Transvaginal Ultrasound
 - if bHCG is > 1200 and there is no intra-uterine pregnancy = probable ectopic
 - An awareness of the limitations of US is as follows:
 - Cardioactivity needs to be seen to confirm intra-uterine pregnancy
 - Cardioactivity can be seen at gestational age **6-6.5 weeks**.
 - Cardioactivity does not exclude ectopic pregnancy in patients undergoing fertility treatment who are at risk of a **heterotopic pregnancy**.
 - Absence of an intrauterine pregnancy translates to a risk of ectopic of about 36%.

III. ANTEPARTUM HAEMORRHAGE

- Vaginal bleeding occurring from the 24th week of pregnancy and prior to the birth of the baby is termed antepartum haemorrhage (APH).

1. PLACENTA PRAEVIA

- Placenta praevia occurs when the placenta is implanted wholly or in part into the lower segment of the uterus.
- If the cervical os is completely covered it is considered a **major praevia (complete)** and if not, then it is considered a **minor praevia (marginal)**.

Placenta praevia



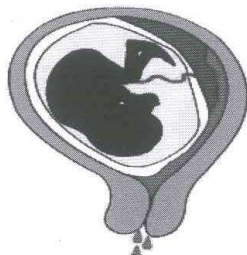
- **Presentation:**
 - **Painless haemorrhage** or **foetal malpresentation** in late pregnancy are classical signs.
 - Abdominal pain can also occur
- **MANAGEMENT:**
 - Antenatal screening at 20 weeks enables detection and expectant management
 - Women who have had a bleed will be managed as in patients from 34 weeks.
 - Asymptomatic women may be managed as outpatients with close monitoring.
 - It is rare to have an undiagnosed placenta praevia present to the ED.

2. PLACENTAL ABRUPTION

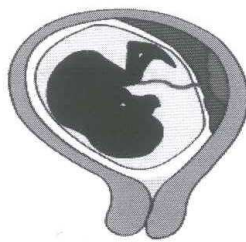
- Placental abruption is the complete or partial premature separation of a normally implanted placenta from the uterus causing haemorrhage into the basalis decidua.
- **RISK FACTORS:**
 - Increased maternal age,
 - Smoking,
 - Use of cocaine,
 - Hypertension,
 - Multiple pregnancy,
 - High parity,
 - Prolonged rupture of membranes and trauma are all associated.
- The primary cause for abruption remains unknown except in cases of trauma.
- **Clinical:**
 - **Fundal tenderness** is associated with vaginal bleeding.
 - **Bleeding** may be concealed in up to 20%.
 - **Foetal distress** is indicative of abruption
 - **Foetal death** is common where separation is more than 50%.

- o DIC occurs in 10%, which can cause long-term renal failure

Placental abruption



Visible bleeding

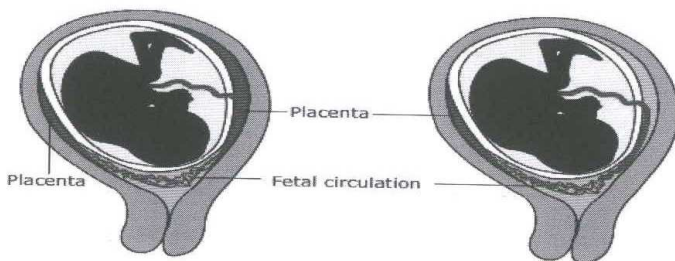


Concealed bleeding

3. VASA PRAEVIA

- Vasa praevia is a condition in which the fetal blood vessels run freely and unsupported through the membranes, over the cervix across the internal os beneath the presenting part, unprotected by placenta or umbilical cord.

Vasa praevia



RISK FACTORS:

- o Placenta praevia,
- o Multilobed placenta,
- o Velamentous insertion of the umbilical cord,
- o Multiple pregnancies
- o IVF pregnancies
- The fetal blood vessels may be ruptured at amniotomy, spontaneous rupture of membranes or during cervical dilatation
- **Clinical:**
 - o **Painless PV bleeding** and **foetal heart activity abnormalities** are common.
 - o Pulsating vessels on vaginal examination are indicative; however PV examination is normally contraindicated because of the possibility of placenta praevia.

4. PLACENTA ACCRETA

- Placenta accreta is abnormal adherence of the placenta to the uterus.
- It is strongly associated with previous caesarean sections and can be identified on U/S.
- It is usually a post partum phenomenon although rarely may cause spontaneous uterine rupture and massive intraperitoneal haemorrhage.

IV. FETO-MATERNAL HAEMORRHAGE (FMH)

- It is most common in the third trimester, during childbirth and following events associated with FMH.
- Such events include **medical interventions** (Chorionic villus sampling, amniocentesis, external cephalic version), **terminations, late miscarriages, APH and abdominal trauma**
- Can occur in the absence of an observed potentially sensitising event
- Causes alloimmunisation
- **Sensitisation:**
 - It has no effect on the mother and usually no adverse effect on the fetus in the primary pregnancy during which it occurs.
 - It is dependent on the volume of fetal blood entering the maternal circulation and the volume of the mother's immune response.
 - It is greatest with the first pregnancy (with the same father) and reduced with subsequent pregnancies
 - Once occurred, **is irreversible**
- **The immune response is:**
 - Usually not detected in the first pregnancy
 - Faster and greater in subsequent pregnancies
 - Causes **fetal anaemia** which in utero leads to **heart failure, hydrops foetalis** and **IUD**.
 - **Neonatal haemolytic disease** of the newborn ensues causing **kernicterus**

CLINICAL ASSESSMENT

- A multidisciplinary approach to assessment and intervention of the shocked pregnant women is required.
- **History**
 - When possible take a full history. Establish why the patient has attended the ED.
 - Pertinent questions include LMP, parity, gravity and outcome of previous pregnancies not resulting in a live birth, paternity of previous pregnancies, rhesus status, sexual history, contraceptive history, fertility treatment, and pelvic surgery.
- **Ask about:**
 - Bleeding amount, colour and consistency and any previous bleeding in this or previous pregnancies
 - Scans in this pregnancy
 - Trauma
 - Pain location, nature and radiation
- **Establish if the patient is shocked:**
 - RR, Sats, HR, BP, CRT, Urine Output
- **Essential investigations**
 - **Urine+/-serum hCG**
 - **FBC, U&E, Clotting studies, G&S +/- Cross match** (at least 4 units if bleeding is heavy), Blood grouping
 - If gestation greater than 20/40, **Consider Kleihauer** (a blood test used to measure the amount of fetal hemoglobin transferred from a fetus to a mother's bloodstream), this determines the need for additional anti-D.
 - Consider **ECG**

- o Not required by: Individuals who are already sensitised are identified though an **indirect Coombs test**.

CLINICAL EXAMINATION

- o Look for evidence of abdominal trauma
- o Estimate PV loss as appropriate to the history
- o **Do not** perform a vaginal examination in women presenting with PV bleeding after the 24th week as this can precipitate catastrophic haemorrhage in undiagnosed placenta praevia.
- o The need for speculum examination should be considered on a case-by-case basis and should only be performed by a clinician competent in the technique.
- o **Use of Doppler and US**
 - The fetal heart is audible with a Doppler probe from 10 weeks.
 - Ongoing fetal monitoring should be by CTG.
 - In the case of abdominal trauma, this should be prolonged monitoring, directed by local guidelines.
 - Increasing availability of US in EDs should enable a rapid scan to be performed by a competent clinician.

ED MANAGEMENT OF BLEEDING IN PREGNANCY

- o **ABC DEFG**
- o Oxygen \pm airway management as appropriate
- o IV access (2 wide bore cannulae) and volume replacement with crystalloid or colloid and blood
- o **Left uterine displacement** can increase cardiac output by 30%
- o Correction of coagulopathy
- o Consider central venous catheterisation both for monitoring and access
- o **Catheterisation.**
- o **Analgesia**
- **Suspected ectopic pregnancy:** definitive management by the gynaecology team.
- **Suspected cervical shock:** remove products of conception from the os with the aid of a speculum and sponge forceps.
- **Continued haemorrhage:** consider administration of **ergometrine** and **oxytocin**.
- **Delivery of the baby:** in severe APH where fetal heart activity is detected, caesarean delivery of the baby should proceed. Where no fetal activity is identified vaginal delivery is advocated.
- **Administration of anti-D** to rhesus-negative women may not always be required.
 - o In these circumstances, the dose to administer is:
 - **Before 20 weeks:** 250 IU IMI to the Deltoid muscle
 - **After 20 weeks:** 500 IU IMI to the Deltoid muscle
 - o After 20 weeks gestation a **Kleihauer test** should be performed to establish the size of the FMH and additional anti-D given as required. This would not be done in the ED.
 - o As anti-D immunoglobulin is a **blood product** there will be a small number of patients with particular religious beliefs to whom this treatment is unacceptable.
 - o *There is no passive immunisation and no alternative treatment.*

V. POST PARTUM HAEMORRHAGE

- PPH is a bleeding of >500 ml in first 24h from delivery. It is described as primary or secondary:
 - **Primary PPH** occurs in the first 24 hours after delivery (also called early PPH) and
 - **Secondary PPH** occurs 24 hours to 12 weeks after delivery (also called late or delayed PPH).
 - **It is normal to expect 200-300ml of blood loss**
 - PPH >500ml
 - Severe PPH (SPPH) >1000ml
 - Life-threatening > 2500ml/40% total blood volume
 - **Causes of Postpartum Hemorrhage: "Four Ts"**

FOUR TS	CAUSE
Tone	Atonic uterus
Trauma	Lacerations, Episiotomy, Hematomas, Inversion, Rupture
Tissue	Retained Placenta or products of conception, Invasive Placenta
Thrombin	Coagulopathies

• RISK FACTORS

Thrombin	<ul style="list-style-type: none"> • Pre-eclampsia • Placenta abruption 	<ul style="list-style-type: none"> • Pyrexia in labour • Bleeding disorders: Haemophilia, anticoagulation, Von Willebrand
Tissue	<ul style="list-style-type: none"> • Retained placenta • Placenta accrete 	<ul style="list-style-type: none"> • Retained products of conception
Tone	<ul style="list-style-type: none"> • Placenta praevia • Previous PPH 	<ul style="list-style-type: none"> • Overdistension of the uterus: Multiple pregnancy, polyhydramnios, macrosomia
Trauma	<ul style="list-style-type: none"> • Caesarean section • Episiotomy 	<ul style="list-style-type: none"> • Macrosomia
Other	<ul style="list-style-type: none"> • Asian ethnicity • Anaemia • Induction 	<ul style="list-style-type: none"> • BMI >35 • Prolonged labour • Age

PRIMARY POSTPARTUM HAEMORRHAGE

- Primary PPH tends to be more severe than secondary PPH and is an obstetric emergency so call seniors immediately.
- **ACTIVE MANAGEMENT**
 - **Uterotonics** such as **oxytocin** reduces the risk of PPH by 60% when given prophylactically. (Syntocinon = synthetic oxytocin and is contra-indicated in patients with hypertension). Carbetocin is used to prevent PPH in caesarean delivery
 - Early clamping of the umbilical cord
 - Controlled traction of the placenta
- In a homebirth setting or where uterotropics are not available, **Misoprostol** (synthetic Prostaglandin E1) may be given to encourage uterine contraction.

ED MANAGEMENT OF POSTPARTUM HAEMORRHAGE

1. PRIMARY PPH

- The patient has lost **500-1000ml blood** and has no clinical features of shock
- **ABC:** oxygen and IV access
- Clinical examination for the cause of haemorrhage
- **Investigations:** FBC, coagulation screen, U&Es, group and save / cross match
- In most cases of primary PPH, uterine atony is the cause. To manage this:
 - **Bimanual uterine compression** and **fundal massage** stimulates contractions
 - Ensure the bladder is empty
 - **Administer Syntocinon** (synthetic oxytocin) (5 units I.V), **Ergometrine** (0.5mg I.V/I.M) and **Carboprost/Haemabate** (synthetic PGF₂) (0.25mg I.M)
 - **1000micrograms misoprostol** (rectal) may also be used if it is suspected that uterine atony is the underlying cause.
 - **Hayman sutures** encourage uterine tone.
- In cases of severe PPH, **uterine artery embolisation** is the suggested method of treatment (Soncini et al, 2007) in both primary and secondary PPH.
- **What about tranexamic acid?** RCOG rarely values the use of tranexamic acid in obstetric haemorrhage.

2. SECONDARY PPH

- Secondary PPH occurs between **24h and 12 weeks** after delivery.
- Bleeding is less severe than in primary PPH.
- The cause is often **uterine atony** or **retained products of conception**.
- Secondary PPH commonly presents to primary care where a full obstetric and haematological history should be obtained.
- **INVESTIGATIONS:**
 - FBC
 - Blood cultures
 - Midstream Urine
 - High vaginal swab
 - Ultrasound can also be used to detect retained products of conception
- Long and complicated labour increase the risk of translocation of flora. **Group B Streptococcus (gram +ve) organisms** often cause **endometritis**.
- Endometritis is often polymicrobial and if endometritis is suspected then broad-spectrum antibiotics are required.
 - In a primary care setting, **amoxicillin** or **co-amoxiclav** is indicated
 - In a secondary care setting, **ampicillin** or **clindamycin** and **metronidazole** is recommended (RCOG, 2009). **Gentamycin** is recommended in more severe cases.
- **COMPLICATIONS OF PPH**
 - *Sequelae of hypovolaemia (shock, renal failure)*
 - *DIC/Sepsis*
 - *Transfusion or anaesthetic reaction*
 - *Fluid overload (pulmonary oedema)*
 - *DVT, VTE*
 - *Anaemia (normocytic normochromic)*
 - **Sheehan syndrome** (postpartum hypopituitarism from pituitary necrosis) which can present as failure to lactate.

VI. PREGNANCY AND TRAUMA

- ATLS approach (primary and secondary survey) including safe transport to trauma centre with obstetric care.
- 80% of women who survive haemorrhagic shock experience fetal death
- **Additional issues:** Anatomical and Physiological changes of pregnancy, Pregnancy specific complications and Foetal issues

1. ANATOMICAL AND PHYSIOLOGICAL CHANGES IN PREGNANCY

- **Airway**
 - **Aspiration risk**
 - **Potentially difficult intubation** (narrow airway, oedematous, bleeds easily)
 - May require: RSI with skilled staff, in-line stabilisation and have DA equipment
- **Breathing**
 - **Smaller FRC** due to gravid uterus, high O₂ consumption -> decreased apnoeic supply
 - **Physiological respiratory alkalosis**, with metabolic acidosis -> a PaCO₂ of 35-40 may already indicate respiratory failure
 - **Give additional high flow oxygen** – target higher SaO₂ due to fetal requirements
 - If intubated – controlled ventilation
 - Fetus may not tolerate permissive hypercapnoea due to increasing acidosis
- **Circulation**
 - **Physiologically lower SBP and DBP**, lower SVR, increased HR and increased CO – must be taken into account on evaluation
 - **Physiologic anaemia and increased blood volume** – may lose 1.2 – 1.5 L of blood volume before showing signs of hypovolaemia
 - **Avoid aortocaval compression syndrome** – keep in Left lateral position or manually displace the uterus
 - **Rh compatible transfusions.** Rh neg mothers will need **Ig** for the immunological risk of fetomaternal haemorrhage -> – **Rh Ig for all Rh D negative women within 72 hours**
 - **ECG changes:** Left axis deviation, flat or inverted T waves, ectopics
 - FAST scan/USS can be difficult c/o gravid uterus
 - **Give blood early**
- **Others**
 - **Radiology:** Foetal exposure but do what needs to be done (more care in first trimester)
 - Uterus is extrapelvic from week 12/Cephalad movement of bowel

2. PREGNANCY SPECIFIC ISSUES

- **Monitor baby (CTG)**
- **Place chest drains slightly higher than normal** c/o Cephalad movement of diaphragm
- **Kleihauer-Betke test** can detect fetal blood in maternal circulation
- **Look for:** retroperitoneal haemorrhage / placental abruption / foetal distress / premature labour / AFE / DIC / uterine rupture
- Pelvic binders in pelvic fracture may be unsuitable

3. FOETAL ISSUES

- **Call for help early** – O&G, Paeds and Anaesthetics
- **Continuous foetal monitoring with CTG** (depending on gestational age > 20 weeks)
- **Intrauterine resuscitation:** foetal oxygenation dependent on mothers oxygenation/ventilation and CO / uterine perfusion
- Maternal compensation for blood loss is at the expense of uteroplacental flow

VII. ABNORMAL VAGINAL BLEEDING

INTRODUCTION

- Abnormal uterine bleeding (formerly, **Dysfunctional Uterine Bleeding [DUB]**) is irregular uterine bleeding that occurs in the absence of recognizable pelvic pathology, general medical disease, or pregnancy.
- It reflects a disruption in the normal cyclic pattern of ovulatory hormonal stimulation to the endometrial lining.
- The bleeding is unpredictable in many ways. It may be excessively heavy or light and may be prolonged, frequent, or random.
- There is a large differential diagnosis in women with abnormal vaginal bleeding.
- A careful menstrual history should be taken, including any history of post-coital or intermenstrual bleeding.
- A pregnancy test must always be performed, regardless of whether the patient has missed a period or not.

VAGINAL BLEEDING TERMINOLOGY

- **Dysmenorrhea:** Painful cramps during menstruation. **Primary dysmenorrhea** is caused by menstruation itself. **Secondary dysmenorrhea** is triggered by another condition, such as endometriosis or uterine fibroids.
- **Amenorrhea:** Absence of menstruation. **Primary amenorrhea** is considered when a girl does not begin to menstruate by the age of 16. **Secondary amenorrhea** occurs when periods that were previously regular stop for at least 3 months.
- **Oligomenorrhea:** Menstrual bleeding with intervals of greater than 35 days.
- **Polymenorrhea:** Menstrual bleeding with intervals of less than 21 days.
- **Menorrhagia:** Menstrual bleeding with excessive flow or duration. Intervals are regular.
- **Metrorrhagia:** Irregular menstrual bleeding.
- **Menometrorrhagia:** Menstrual bleeding with excessive flow or duration. Intervals are irregular.
- **Intermenstrual bleeding:** Variable amounts of bleeding between normal regular menstrual periods.
- **Heavy menstrual bleeding** is both menorrhagia and menometrorrhagia, and refers to the menstrual blood loss of higher than 80 ml per month.

1. MENORRHAGIA

- Menorrhagia is excessive menstrual blood loss.
- The differential includes:
 - **Dysfunctional uterine bleeding**—heavy or irregular periods without obvious pelvic pathology. Often seen around menarche due to hormonal imbalance. Symptomatic relief with NSAIDs (e.g. mefenamic acid) are the mainstay of treatment.
 - **Fibroids.**
 - **Endometriosis.**
 - **Pelvic inflammatory disease.**
 - **IUCD.**
 - **Polyps.**
 - **Hypothyroidism.**

2. POST-MENOPAUSAL BLEEDING

- Post-menopausal bleeding is one of the most common presentations to a gynaecology clinic. The differential includes:
 - Atrophic vaginitis.
 - Fibroids.
 - Endometrial polyps.
 - Endometrial hyperplasia.
 - Endometrial carcinoma.
 - Cervical carcinoma.
 - Vaginal carcinoma.
 - Bleeding from non-gynaecological sites, e.g. Urethra, Bladder, or Lower GI tract.
- An abdominal examination, speculum, and bimanual vaginal examination should be performed to look for evidence of tenderness or **masses**.
- Patients should be referred to gynaecology as an out-patient for further investigation.

3. VAGINAL BLEEDING UNRELATED TO MENSTRUATION OR PREGNANCY

- **Trauma.**
- **IUCD insertion.**
- **Post-gynaecological operations.**
- **Cervical erosions**—occur when the stratified squamous epithelium is replaced by columnar epithelium. The cervix appears red and the patient may experience post-coital or inter-menstrual bleeding.
- **Cervical polyp.**
- **Cervical cancer**—90% are squamous carcinoma. Speculum examination may reveal nodules, ulcers, or erosions, which may bleed on contact.
- **Endometrial cancer.**
- **Fibroids.**
- **Genital ulcers.**
- **PID.**
- **Bleeding diathesis**, e.g. thrombocytopenia, haemophilia.
- **Anti-coagulant medication.**
- **Oral contraceptive problems**—breakthrough bleeding due to endometrial hyperplasia.
- **ED MANAGEMENT:**
 - Most patients with vaginal bleeding can be managed as outpatients with GP or gynaecology follow up.
 - Patients with evidence of severe bleeding or hypovolaemia should be resuscitated and admitted.
 - Patients with suspected genital tract malignancy should be referred urgently for gynaecology follow up.

VIII. PREECLAMPSIA & ECLAMPSIA

• DEFINITION

- **Preeclampsia** refers to the new onset of **hypertension** and **either proteinuria or end-organ dysfunction or both** after 20 weeks of gestation in a previously normotensive woman.
- **Eclampsia** refers to the development of **grand mal seizures** in a woman with preeclampsia, in the absence of other neurologic conditions that could account for the seizure.

Criteria of Pre-eclampsia

- SBP ≥ 140 mmHg or DBP ≥ 90 mmHg on 2 occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive patient
- If SBP ≥ 160 mmHg or DBP ≥ 110 mmHg, confirmation within minutes is sufficient **and**
- Proteinuria ≥ 0.3 grams in a 24-hour urine specimen or protein (mg/dL)/creatinine (mg/dL) ratio ≥ 0.3
- Dipstick $\geq 1+$ proteinuria if a quantitative measurement is unavailable
- In patients with **new-onset hypertension without proteinuria**, the new onset of any of the following is diagnostic of preeclampsia:
 - Platelet count $< 100,000/\text{microliter}$
 - Serum creatinine > 1.1 mg/dL or doubling of serum creatinine in the absence of other renal disease
 - Liver transaminases **at least twice** the normal concentrations
 - Pulmonary edema
 - Cerebral or visual symptoms
- **Severity of preeclampsia is based on BP measurement alone:**
 - **Mild:** SBP=140 to 149 mmHg and/or DBP=90 to 99 mmHg.
 - **Moderate:** SBP=150 to 159 mmHg and/or DBP=100 to 109 mmHg.
 - **Severe:** SBP is ≥ 160 mmHg and/or DBP ≥ 110 mmHg.

• RISK FACTORS FOR THE DEVELOPMENT OF PRE-ECLAMPSIA

- *First pregnancy*
- *Multiple pregnancy*
- *Obesity*
- *Age older than 35*
- *History of diabetes*
- *History of hypertension*
- *History of kidney disease*

• FEATURES OF SEVERE PRE-ECLAMPSIA

- *Sustained severe hypertension ($> 160/90$ mmHg)*
- *Severe proteinuria*
- *Oliguria (urine output $< 500\text{mls}/24$ hours)*
- *Neurological symptoms and signs e.g. headache, papilloedema, clonus*
- *Platelet count $< 100 \times 10^9/\text{L}$*
- *Epigastric pain and/or right upper quadrant tenderness*
- *Elevated liver enzymes (AST and ALT)*

- **COMPLICATIONS OF PRE-ECLAMPSIA**

- *Eclampsia*
- *HELLP syndrome*
- *Disseminated intravascular coagulation*
- *Renal failure*
- *ARDS: Adult respiratory distress syndrome*
- *Rupture of liver*
- *Stroke*
- *Cerebral haemorrhage/ Cortical blindness*
- *Pulmonary oedema*

- **INVESTIGATIONS FOR PRE-ECLAMPSIA**

- **FBC:** risk of thrombocytopenia and haemoconcentration. Blood film should be checked because the patient may develop microangiopathic haemolytic anaemia.
- **Clotting screen:** should be checked if the patient is thrombocytopenic.
- **Renal function:** risk of renal failure.
- **LFTS:** elevated transaminases in HELLP syndrome.
- **Urinary dipstick:** ≥ 2 + protein indicates significant proteinuria and the need for 24-hour urine collection.
- Foetal monitoring via **ultrasound** and **cardiotocography**

- **MANAGEMENT OF PRE-ECLAMPSIA IN THE ED**

- Upper abdominal pain in pregnancy may indicate pre-eclampsia
- **All women who present with upper abdominal pain and tenderness in pregnancy (usually after 20 weeks' gestation):**
 - **Measure BP:** If $> 140/90$ mmHg seek advice from the obstetric unit in which the woman is booked
 - **Test for proteinuria:** If proteinuria (i.e., more than a trace) is present in an MSU and especially if hypertension is detected refer immediately for admission to the maternity unit. (**Don't take "No" for an answer!!!**)
- Once admitted, blood should be analysed for, among other things, **thrombocytopaenia** and **hepatic dysfunction**.
- If you remain concerned about the epigastric pain and tenderness in the absence of hypertension or proteinuria review the following day.
- Inform the on-call Obstetrics early,
- Move the patient to resuscitation room with full monitoring.
- Consider positioning the patient left lateral.
- Control hypertension with **IV labetalol or hydralazine**.
 - **Hydralazine:** Initial 5-10mg slow bolus; then repeat boluses or infusion 50-100µg/min
 - **Labetolol:** Initial 20-50mg slow bolus; Then infusion 2mg/min, titrated as required
- Careful fluid management is required. Fluid overload is a significant cause of maternal death due to pulmonary oedema. Limit fluids to approximately **1ml/kg/hr**.
- Urine output should be monitored.
- **Magnesium** should be considered in women with **severe pre-eclampsia** (systolic BP ≥ 170 mmHg or diastolic BP ≥ 110 mmHg plus significant proteinuria $> 1\text{g/L}$).
- **Delivery** is the definitive treatment for pre-eclampsia.
- However, 44% of eclampsia occurs post-partum.

• ED MANAGEMENT OF ECLAMPSIA

- ABC approach
- Airway and breathing adequacy should be assessed.
- High-flow supplemental oxygen should be given.
- Ventilation should be assisted if inadequate. **Intubation** should be considered early due to the increased risks of aspiration and ventilatory inadequacy in pregnancy.
- **Magnesium** is the therapy of choice to control seizures. A loading dose of **4 g IV should be given over 5–10 minutes followed by maintenance of 1 g/hour for 24 hours.**
- A further **bolus of 2 g** can be given if the patient has recurrent seizures.

• Management of HTN in Preeclampsia/Eclampsia

- **Hydralazine:** Initial 5–10mg slow bolus; then repeat boluses or infusion 50–100µg/min
- **Labetalol:** Initial 20–50mg slow bolus; Then infusion 2mg/min, titrated as required
- **Nicardipine:** Infusion of 2.5–5mg/hr; Increase to a maximum of 15mg/hr
- *Nitroprusside should be avoided in pregnancy because of its potential toxicity to the foetus.*

MAGNESIUM SULPHATE

• CONTRA-INDICATIONS

- Neuromuscular disease: Myasthenia gravis
- Renal failure,
- Cardiac disease

• INDICATIONS

- **Eclampsia-** Magnesium Sulphate rarely required to stop fit – usually self-limiting
- **Severe pre-eclampsia** where the decision to deliver has been made and where there is one other of the following criteria:
 - Hypertension with diastolic BP ≥ 110 mm Hg or systolic BP 170 mm Hg on two occasions and proteinuria $\geq 3+$
 - Hypertension with diastolic BP ≥ 100 mg Hg or systolic BP ≥ 150 mm Hg on two occasions and proteinuria $\geq 2+$ (0.3 g/day) and at least two of the signs of imminent eclampsia.

• MgSO₄

- **Loading dose: 4 grams I.V. over 5 mins**
- **Maintenance infusion: 1g/hr for at least 24 hours** after the last seizure
- Recurrent seizures should be treated by a further **bolus of 2g**
- **Side effects:**
 - Nausea, vomiting and flushing (use Maxolon)
 - Respiratory arrest
 - Renal failure
 - Hyporeflexia
- **Magnesium toxicity**
 - **Antidote:** Calcium Gluconate 1 gram over 10 mins
 - **Monitor:** reflexes, resps ($>16/\text{min}$), SpO₂, ECG for first hour

• Recurrent seizures after MgSO₄

- Treat with a further bolus of 2g
- RSI with **Thiopentone/ventilation**
- Treat hypertension (MgSO₄ may reduce BP otherwise, give **Hydralazine**)

IX. HELLP SYNDROME

• OVERVIEW

- HELLP syndrome, named for 3 features of the disease (Hemolysis, Elevated Liver enzyme levels, and Low Platelet levels), is a life-threatening condition that can potentially complicate pregnancy.
- The cause of HELLP syndrome is currently unknown, although theories as described in Pathophysiology have been proposed.

• RISK FACTORS FOR HELLP SYNDROME

- *Maternal age older than 34 years*
- *Multiparity*
- *White race or European descent*
- *History of poor pregnancy*

CLINICAL FEATURES

• History

- No 'typical' clinical symptoms
- Epigastric or RUQ pain
- Weight gain (oedema)

• Examination

- Hypertension
- Tender RUQ
- Oedema
- Polyuria from nephrogenic Diabetes Insipidus

• INVESTIGATIONS

- Microangiopathic haemolytic anaemia (MAHA)
- Elevated LFT's – bilirubin, AST, ALT, LDH
- Low platelets
- Normal PT, APTT and coagulation screen
- Haemolysis on blood film
- Haptoglobins: low

• COMPLICATIONS OF HELLP SYNDROME

- **Haemorrhage**
 - *Abruptio placentae*
 - *Severe post partum haemorrhage*
 - *Subcapsular liver haematoma*
 - *Intracerebral or brainstem haemorrhage*
 - *DIC*
- **Infarction**
 - *Liver infarct/ Cerebral infarct*
- **Pregnancy**
 - *Overlap with preeclampsia*
 - *Preterm delivery*
 - *IUFD*
- **Other**
 - *Visual impairment due to retinopathy*
 - *Pulmonary oedema – higher risk in post partum onset of HELLP*
 - *Acute kidney injury – higher risk in post partum onset of HELLP*

DIFFERENTIAL DIAGNOSIS

- Pre-eclampsia / eclampsia
- Acute fatty liver of pregnancy
- Acute hepatitis
- Hemolytic-uremic syndrome (HUS)
- Thrombotic Thrombocytopenic Purpura (TTP/rare in pregnancy)
- Immune Thrombocytopenic Purpura (ITP)
- DIC (e.g. from PPH or amniotic fluid embolism)
- Other causes of haemolysis (e.g. AIHA, sepsis)
- Other causes of acute abdomen

ED MANAGEMENT OF HELLP SYNDROME

Resuscitation

- Prepare for major haemorrhage
- Major life threats are hepatic hemorrhage, subcapsular hematoma, liver rupture, and multi-organ failure

Specific treatment

- **Delivery** is indicated if the HELLP syndrome occurs after the 34th gestational week or the foetal and/or maternal conditions deteriorate.
- **Seek and treat complications** (e.g. APO, DIC, MODS)
- **Anti-hypertensives** to keep BP below 155/105 mmHg
 - Labetolol or Hydralazine or Nifedipine
- **MgSO₄ IV** for eclamptic seizure prophylaxis
- **Corticosteroids (IV) for Lung maturity**
 - No clear benefit for HELLP per se
 - Given for fetal lung maturity from 24 to 34 weeks: either 2 doses of 12 mg betamethasone 24 hours apart or 6 mg dexamethasone 12 hours apart before delivery.
- **Liver haemorrhage**
 - Manage conservatively where possible
 - Correct coagulopathy
 - Surgery includes drainage of the hematoma, packing, oversewing of lacerations, or partial hepatectomy
 - Consider arterial embolisation
- **Exchange transfusion**
 - Considered in situations of progressive elevation of bilirubin or falling Hb or PLTs and ongoing deterioration in maternal condition.

Supportive care and monitoring

- Consider invasive monitoring

Disposition

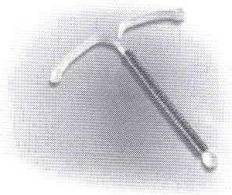
- OT or HDU/ ICU setting
- Consider transfer to a liver transplant center

X. EMERGENCY CONTRACEPTION

- Women requesting emergency contraception have 3 choices:

1. LEVONELLE 1.5mg

- This is levonorgestrel and is licensed up to **72 hours** after UPSI (Unprotected Sexual Intercourse). If vomiting occurs **within 2 hours of ingestion**, another tablet should be given. It works mainly by inhibiting ovulation.



2. ULIPRISTAL ACETATE

- This is the newest treatment available and is licensed up to **120 hours after UPSI**.
- If vomiting occurs within **3 hours of ingestion** another tablet should be given.
- It also works mainly by inhibiting ovulation.
- It should be avoided in patients taking **enzyme-inducing drugs**, severe hepatic impairment or severe asthma that requires oral steroids.
- Levonelle and ulipristal are less effective in women with higher BMIs.
- **Missed regular Oral contraception Pill:** If one pill has been missed (48-72 hours since last pill in current packet or 24-48 hours late starting first pill in new packet) then the following contraceptive cover is required:
 - The missed pill should be taken as soon as possible
 - The remaining pills should be continued at the usual time
 - Emergency contraception is not usually required but may need to be considered if pills have been missed earlier in the packet or in the last week of the previous packet.

3. COPPER IUD

- This can be fitted up to **5 days after UPSI or ovulation**, whichever is longer.
- Failure rate is less than 1 in a 1000, making it 10-20 times more effective than oral emergency contraceptive options.

4. THE FRASER GUIDELINES (GILICK COMPETENCE)

- Lord Fraser stated that a Doctor could proceed to give advice and treatment:
 - **"Provided he is satisfied in the following criteria:**
 - That the girl (although under the age of 16 years of age) will understand his advice;
 - That he cannot persuade her to inform her parents or to allow him to inform the parents that she is seeking contraceptive advice;
 - That she is very likely to continue having sexual intercourse with or without contraceptive treatment;
 - That unless she receives contraceptive advice or treatment her physical or mental health or both are likely to suffer;
 - That her best interests require him to give her contraceptive advice, treatment or both without the parental consent." (Gillick v West Norfolk, 1985)

CHAPTER 28. TOXICOLOGY & EMERGENCY

I. CARBON MONOXIDE POISONING

OVERVIEW

- Carbon monoxide (CO) is a colourless, odourless gas produced by incomplete combustion of carbonaceous material. CO poisoning may be acute or chronic.
- Exposure is most commonly from suicide attempts using car exhaust, and accidental exposures from incomplete combustion in charcoal burners, faulty heaters, fires, and industrial accidents.
- Chronic CO poisoning may have an insidious presentation (e.g. intermittent headaches), and a high index of suspicion is required in at-risk groups (e.g. fires inside the home)

TOXICODYNAMICS

- Carbon monoxide has **~210 times** the affinity for haemoglobin than oxygen.
- Binding therefore renders haemoglobin oxygen carrying capacity and delivery to the tissues. This can result in tissue **hypoxia and ischaemic injury**.
- CO also binds to intracellular cytochromes, impairing aerobic metabolism
- Typical clinical symptoms and signs relative to COHb (Normal = 0.5%):**
 - <10%:** Nil, commonly found in smokers
 - 10 – 20%:** Nil or vague nondescript symptoms
 - 30 – 40%:** Headache, tachycardia, confusion, weakness, nausea, vomiting, collapse
 - 50 – 60%:** Coma, convulsions, Cheyne-Stokes breathing, arrhythmias, ECG changes
 - 70 – 80%:** Circulatory and ventilatory failure, cardiac arrest, Death

CLINICAL FEATURES

- Acute poisoning**
 - The **cherry red skin** colour produced when carboxyhaemoglobin (COHb) concentrations exceed about 20% is rarely seen in life.
 - CNS:** Headache, Nausea, Dizziness, Confusion, Mini Mental Status Examination Errors, Incoordination, Ataxia, Seizures and finally Coma.
 - CVS:** Dysrhythmias, Ischaemia, hyper or hypotension (exacerbated in patients with anaemia or underlying cardiovascular disease)
 - GI:** Abdominal Pain, N+V, Diarrhoea
 - RESP:** Dyspnea, Tachypnea, Chest Pain, Palpitation
 - Other:**
 - Non-cardiogenic pulmonary oedema
 - Lactic acidosis
 - Rhabdomyolysis
 - Hyperglycaemia
 - Disseminated intravascular coagulation
 - Bullae
 - Alopecia
 - Sweat gland necrosis
- Chronic exposures**
 - May have similar effects to acute poisoning, but often with a gradual, insidious onset, and symptoms may fluctuate with varying levels of exposure to CO over time.

- o Compared with acute exposures, they typically involve a lower dose of carbon monoxide for a long period, which increases the risk of developing neurological complications.
- o Symptoms are usually non-specific but can include Headache, Personality changes, Poor Concentration, Dementia, Psychosis, Parkinsonism, Ataxia, Peripheral Neuropathy and Hearing loss

INVESTIGATIONS

• Bedside

o ABG

- HbCO: Elevated levels are significant, but low levels do not rule out exposure.
- Lactate (Tissue Hypoxia)
- PaO₂ should be normal, SpO₂ only accurate if measured (not calculated from PaO₂)
- MetHb (exclude)

o ECG: Sinus Tachycardia, Ischaemia

o Urinalysis: Positive for albumin and glucose in chronic intoxication; β -HCG for pregnancy.

• Laboratory

o FBC (Mild Leukocytosis)

o BSL (Hyperglycaemia)

o U&E (Hypokalaemia, Acute renal failure from myoglobinuria)

o CK (Rhabdomyolysis)

o LFT derangement (ischaemia)

o Ethanol level (Polypharmacy OD)/ Cyanide level (Industrial fire, Cyanide exposure)

• Imaging

o CT/MRI brain: may demonstrate cerebral oedema, cerebral atrophy, basal ganglia injury or cortical demyelination

o CXR: pulmonary symptoms

ED MANAGEMENT OF CO POISONING

• Resuscitation

- o FiO₂ 1.0 (continue until patient asymptomatic or CO level < 10%)
- o Cardiac monitoring
- o Intubate the comatose patient

• Specific Treatment

o High flow O₂ via non-rebreather mask until asymptomatic

- Or for 24 hours while fetal wellbeing is assessed if pregnant

o Hyperbaric oxygen (HBO)

- Role is uncertain
- 3 atmospheres will decrease the half life of carboxyHb from 6 hours to ~ 24 minutes

▪ INDICATIONS OF HBO:

- All pregnant patients
- Significant LOC
- Signs of ischaemia
- Significant neurological deficit
- Metabolic acidosis

CONTRA-INDICATIONS OF HBO

- Chest trauma
- Serious drug overdose,
- Severe burns
- Uncooperative patient

COMPLICATIONS OF HBO

- Decompression sickness
- Rupture of tympanic membranes
- Damaged sinuses
- Oxygen toxicity
- Problems due to lack of monitoring

- o Supportive care and monitoring
- o Seek and treat cause and complications
 - Address suicidality if present
 - Treat coexistent cyanide toxicity if suspected (e.g. House fire)
 - Seek and treat ischaemic complications and neurological sequelae

DISPOSITION

- o Depending on severity:
 - Home, ward environment, ICU and/or hyperbaric chamber
- o Consider transfer to hyperbaric facility if severe intoxication or persistent symptoms after 4h
- o Suicidality requires a psychiatric referral/ admission
- o Work or home environment assessment
- o Check if other household members are affected
- o **Follow up**
 - Anyone with a neurological deficit will require **neuropsychiatric testing in 1-2 months**
 - Complications are present in 30% of survivors at 1 month and 6-10% at 12 months

PREGNANCY

- Significant CO poisoning in the mother often results in foetal death or neurological damage
- The foetus is thought to be especially susceptible to CO poisoning due to:
 - o Low oxygen pressures
 - o High affinity of foetal haemoglobin for CO
 - o Much longer half-life of CO in the foetal circulation
- **There may be an added benefit from HBO in this setting**
 - o HBO shortens the half-life of CO
 - o Allows delivery of oxygen to the tissues independent of haemoglobin
 - o HBO appears to be safe in pregnancy

II. CYANIDE POISONING

- A 37 year-old man is BIBA to the Emergency Department following a fire at his apartment. He has a fluctuating level of consciousness (currently GCS 11) and is hypotensive (BP 85/50). He has no evidence of airway compromise, burns or other significant injury.
- A venous gas shows that he has a **COHb of 21%** and a **Lactate of 14 mmol/L**.
- Can you keep this man alive?



Q1. What is the likely diagnosis and what is the significance of the lactate of 14 mmol/L in this setting?

- **Smoke inhalation** resulting in **Cyanide** and **Carbon Monoxide** poisoning.
- *In the absence of severe burns, there is a strong correlation between the severity of cyanide intoxication and serum lactate. For instance, a serum lactate > 10 mmol/L predicts a cyanide level >40 micromol/L with a sensitivity of 87% and a specificity of 94%.*
- Cyanide levels help confirm the diagnosis in retrospect (take blood in a heparinised tube):
 - >20 microM: symptomatic
 - >40 microM: potentially toxic
 - >100 microM: lethal

Q2. What is the the significance of the COHb of 21%?

- An elevated carboxyhemoglobin level confirms the diagnosis of **carbon monoxide toxicity**, but the level only loosely correlates with the severity of symptoms following exposure.
- Smokers may have a baseline COHb level up to about 10%. A general guide to the clinical features at different levels is:
 - 10% — Asymptomatic or Mild Headache
 - 20% — Dizziness, Nausea, Dyspnea, Throbbing Headache
 - 30% — Vertigo, Ataxia, Altered Vision
 - 40% — Confusion, Coma, Seizures, Syncope
 - 50% — Arrhythmias, Seizures, Cardiorespiratory Arrest

Q3. What are the mechanisms of toxicity for these agents?

- **Carbon monoxide**
 - Binds hemoglobin with >200 times the affinity of oxygen, resulting in 'anemic hypoxia' (reduced ability of the blood to carry oxygen).
 - Binds to intracellular cytochromes and myoglobin, which contributes to 'histotoxic hypoxia' (reduced ability of the blood to utilise oxygen).
 - Initiates endothelial oxidative injury, lipid peroxidation and an inflammatory cascade (probably an important contributor to delayed neuropsychiatric sequelae).

- **Cyanide**
 - Binds the ferric (Fe^{3+}) ion of cytochrome oxidase causing 'histotoxic hypoxia' and lactic acidosis.
 - Stimulates biogenic amine release causing **pulmonary and coronary vasoconstriction**.
 - Stimulates neurotransmitter release, such as N-methyl-D-aspartate (NMDA), causing **neurotoxicity and seizures**.

Q4 What specific treatment should be given for the COHb of 21%?

- **Oxygen!**
 - In the first instance, **high flow oxygen via a non-rebreather mask** (as close to 100% oxygen as possible) should be administered (unless intubation and ventilation is indicated). The administration of oxygen enhances the elimination of CO, which depends on the dissolved oxygen tension in the blood.
 - Approximate elimination half-lives of CO when treated with:
 - **Room air** — 240 min,
 - **100% oxygen** — 90 min
 - **Hyperbaric oxygen (100% oxygen at 3 atmospheres)** — 23 minutes
- **Hyperbaric oxygen**
 - Should always be considered in patients with risk factors for neuropsychiatric sequelae and in the pregnant patient.
 - However, despite a number of trials the indications and effectiveness of this therapy are unclear. In critically ill patients the administration of hyperbaric oxygen may present major logistical problems.
- Oxygen should be administered until all symptoms have resolved.

Q5. What treatment(s) should be considered for the likely cause of the hyperlactemia?

- The relative efficacy of different cyanide antidotes is not well defined.
- However, most authorities recommend administration of an antidote if cyanide poisoning is suspected and there evidence of serious clinical toxicity (i.e. altered mental status, seizures, hypotension or metabolic acidosis).
- The main cyanide antidotes that may be given in the emergency department are:
 - **Dicobalt Edetate and Hydroxocobalamin**
 - **Sodium Thiosulfate**
 - **Amyl nitrite (inhaled), Sodium nitrite (IV) and Dimethyl aminophenol (IV/IM)** convert hemoglobin (Fe^{2+}) to methemoglobin (Fe^{3+}) which binds cyanide forming cyanhemoglobin.

Q6. What is the patient's prognosis?

- **Deaths** from both cyanide toxicity and CO poisoning **tend to occur rapidly**, prior to arrival at an ED. For those that arrive at hospital alive survival is likely even if only oxygen and meticulous supportive care is provided. Depending on the duration and severity of tissue hypoxia, multiple organ dysfunction syndrome (MODS) may be the patient's recovery.
- The patient is at risk of **longer term neuropsychiatric sequelae**.
 - Loss of consciousness, persistent neurological dysfunction and metabolic acidosis are all high-risk features for neuropsychiatric sequelae in the context of CO poisoning (other risk factors include age >55 years and cerebellar signs — although in this case the picture is further complicated by the co-existence of cyanide toxicity).
 - Appropriate follow up is necessary, and in severe cases an MRI may show evidence of demyelination, basal ganglia injury, cerebral edema or atrophy.
 - Similar longterm complications may occur as a result of severe cyanide poisoning.

III. TRICYCLIC ANTIDEPRESSANT OVERDOSE

- Any overdose of amitriptyline **> 10 mg/kg** is potentially life-threatening.
- An overdose **> 30 mg/kg** will result in severe toxicity, cardiotoxicity and coma.
- The toxic effects of TCAs are mediated by several pharmacological effects:
 - Anticholinergic effects*
 - Direct alpha adrenergic blockade*
 - Blockade of noradrenaline reuptake at the preganglionic synapse*
 - Blockade of sodium channels*
 - Blockade of potassium channels*

CLINICAL EFFECTS

Anticholinergic	<ul style="list-style-type: none"> Dry mouth, Dry Skin Constipation, Urinary retention Mydriasis/Blurred vision Aggravation narrow angle glaucoma
Anti-alpha adrenergic	<ul style="list-style-type: none"> Orthostatic hypotension
Antihistaminic	<ul style="list-style-type: none"> Sedation
Cardiac	<ul style="list-style-type: none"> Tachycardia, Hypotension Palpitation, Chest pain.
CNS	<ul style="list-style-type: none"> Decrease mental status, Respiratory depression, Drowsiness, Confusion, Convulsion, Coma.

- The cardiotoxic effects of TCAs are mediated by the blockade of **Na channels**, which causes QRS broadening, and blockade of **K channels**, which causes QT interval prolongation.
- The degree of QRS broadening correlates with adverse events:**
 - QRS > 100 ms** is predictive of **seizures**
 - QRS > 160 ms** is predictive of **ventricular arrhythmias**
- The ECG changes seen in TCA overdose include:**
 - Sinus tachycardia (very common)
 - Prolongation of the PR interval & Broadening of QRS complex
 - Prolongation of the QT interval & Ventricular arrhythmias (severe toxicity)

MANAGEMENT OF TCAs POISONING IN THE ED

- Clear airway and consider early intubation
- Administer high flow **oxygen** and **attach monitoring equipment**
- Secure **IV access** (Blood for ABG, U&E, Paracetamol level...),
- Activate charcoal 1 g/kg PO/NG** (can be given if presents within first hour)
- Avoid antiarrhythmics** as may worsen hypotension and conduction abnormalities
- Administer **NaHCO₃ 8.4% 50-100mls IV** as the first-line drug treatment for arrhythmias
- Lidocaine 1.5 mg/kg IV** can be given as a second-line drug for arrhythmias
- Magnesium sulphate 2g IV over 30 minutes** may also be helpful
- Glucagon 1mg IV** may help severe hypotension
- Administer **Lorazepam 4mg IV** for seizures
- Hypertonic saline 10mL/kg** (of 7.5% saline) may be beneficial
- Noradrenaline 0.02-1.5 mcg/kg/min IV** infusion for severe hypotension
- Adrenaline 0.02-1.0 mcg/kg/min IV** infusion for severe hypotension

IV. PARACETAMOL OVERDOSE

BACKGROUND

- Paracetamol overdose is the most common overdose in the U.K. and is also the commonest cause of acute liver failure.
- The liver damage is caused by a metabolite of paracetamol, *N*-acetyl-*p*-benzoquinoneimine (NAPQI), which depletes the liver's stores of **Glutathione** and directly damages liver cells.
- An overdose of greater than **12 g or > 150 mg/kg body weight** may cause severe liver damage and death. Acute renal tubular damage and necrosis may also occur.
- **Do NOT take plasma levels within 4 hours of ingestion as they are unreliable.**
- Patients may give inaccurate histories or if there is doubt about the timing or the need for treatment: **treat with NAC.**
- **Methionine** is ineffective in patients who have been given **oral activated charcoal.**
- **NAC** is the treatment of choice when patients are vomiting or present **more than 8 hours after ingestion.**
- **SIGNS AND SYMPTOMS**
 - Initially asymptomatic
 - End-organ toxicity often does not manifest until 24-48 hours after an acute ingestion.
- **Minimum toxic doses:**
 - Adults: 7.5-10 g
 - Children: 150 mg/kg; 200 mg/kg in healthy children aged 1-6 years
- **High risk patients (Risk of Liver damage)**
 - Regular alcohol ingestion
 - Other enzyme (liver microsomal oxidases) inducers (e.g. carbamazepine, phenytoin, phenobarbitone, primidone and rifampicin)
 - Glutathione depletion (e.g. malnutrition and HIV)
- The earliest and most sensitive indicator of liver damage is a **prolonged INR**, which starts to rise at around **24 hours after overdose.**
- **LFTs** are usually normal **until around 16 hours after overdose.**
- **AST and ALT levels** then sharply rise and can reach > 10,000 units/L by **72-96 hours** after overdose.
- **Bilirubin** levels rise more slowly and reach their maximum at around **5 days.**

MANAGEMENT OF PARACETAMOL OD IN THE ED

1. **Management of Adult patients who present within 1-4 hour of ingestion**
 - Consider **charcoal** if more than 150 mg/kg body weight taken, presentation within 1 hour of ingestion and able to control the airway.
 - **Take blood for plasma paracetamol concentration at 4 hours post ingestion.**
 - Assess whether at high risk of severe liver damage (see above).
 - Confirm timings of ingestion.
2. **Management of Adult patients who present within 4-8 hours of ingestion**
 - **Do not** start NAC immediately.
 - Wait until 4 hours post ingestion and take Paracetamol/salicylates levels.
 - Start NAC if level taken at 4 hours is in the appropriate treatment range.
 - If the paracetamol concentration result is not available within 8 hours of ingestion (> 150 mg/kg or > 12 g in total) **start NAC immediately.**

- o It can be stopped later if subsequent level well below treatment line.

3. Mx of all patients who present 8-15 hours after ingestion.

- o Urgent action is required (antidote efficacy drops sharply).
- o **Give NAC immediately** without waiting for the result of the plasma paracetamol concentration measurement if it is thought that more than 150 mg/kg body weight or a total of 12 g or more has been ingested.
- o **Take Paracetamol/Salicylates levels, INR, Creatinine and ALT.**
- o If the paracetamol concentration result is not available within 8 hours of ingestion (> 150 mg/kg or > 12 g in total) **start NAC immediately.**
- o In patients already receiving NAC, only discontinue NAC if the plasma paracetamol concentration is below the treatment line on the graph and there is no abnormality of the INR, plasma creatinine or ALT and the patient is asymptomatic.
- o Continue the infusion if there is any doubt as to the timing of the overdose.
- o At the end of NAC infusion **check INR and plasma creatinine concentration.**
- o Patients who are symptomatic or in whom the INR and/or plasma creatinine are abnormal require further monitoring. **Vitamin K** should be given if the INR is increased.
- o **FFP / clotting factors** are only indicated **for active bleeding.**

4. Mx of patients who present 15-24 hours after ingestion:

- o **Start NAC immediately.**
- o Measure the plasma paracetamol concentration on admission.
- o The infusion may be stopped and the patient discharged from medical care if each of the following criteria is met:
 - The patient is asymptomatic.
 - The INR and plasma creatinine are normal.
 - The plasma paracetamol concentration is **less than 10 mg/L** (0.07 mmol/L) 24 hours after ingestion.
- o Patients in whom the INR and/or plasma creatinine are abnormal or whose plasma paracetamol concentrations exceed 10 mg/L at 24 hours after ingestion require further monitoring and contact with a hepatologist.

5. Mx of patients who present longer than 24 hours after ingestion:

- o All should have their **INR, Plasma Creatinine concentration, ALT and Venous pH** (or hydrogen ion / bicarb concentration) determined.
- o We recommend that they **all** be discussed with a poisons information centre or a specialist liver or poisons unit.

6. Specialist advice on those with liver disease.

- Liver transplantation is occasionally needed for liver failure secondary to paracetamol overdose for patients who presented or were treated late.
- **WHEN TO TRANSFER PATIENTS TO A LIVER TRANSPLANT CENTRE**
 - o *High-risk features mandating admission to a liver transplant centre are:*
 - **INR >3.0 at 48 hours or >4.5 at any time**
 - **Oliguria or creatinine > 200 micromol/L**
 - **Acidosis with pH < 7.3 after resuscitation**
 - **Systolic hypotension with BP < 80mmHg**
 - **Hypoglycaemia**
 - **Severe thrombocytopenia**
 - **Encephalopathy of any degree**

1. N- ACETYL CYSTEINE (NAC)

- **NAC increases Glutathione availability** leading to direct binding to NAPQI.
- NAC is nearly 100% hepatoprotective when it is **given within 8 hours** after an acute acetaminophen ingestion, but can be beneficial in patients who present more than 24 hours after ingestion loading dose of 140 mg/kg.
- Acetylcysteine should be administered by intravenous infusion preferably using Glucose 5% as the infusion fluid. Sodium Chloride 0.9% solution may be used if Glucose 5% is not suitable. The full course of treatment with acetylcysteine comprises of 3 consecutive intravenous infusions.
- **Methionine P.O. 2.5 g 4 hourly to a total dose of 10 g.** is a useful alternative in patients who refuse treatment.
- Doses should be administered sequentially with no break between the infusions.
- The patient should receive a total dose of **300 mg/kg body weight over a 21 hour period** as follows (**Each ampoule = 200mg/mL acetylcysteine**):
- **Adults:**
 - **Loading dose:** 150 mg/kg IV; mix in 200 mL of 5% dextrose in water (D5W) and infuse over 1 h.
 - **Dose 2:** 50 mg/kg IV in 500 mL D5W over 4 h.
 - **Dose 3:** 100 mg/kg IV in 1000 mL D5W over 16 h.
- In patients who weigh more than 100 kg, limited data suggest a loading dose of 15,000 mg infused IV over 1 hours, then a first maintenance dose of 5,000 mg IV over 4 hours and a second maintenance dose of 10,000 mg over 16 hours.

• Children

Table 5: Acetylcysteine administration to children, based on body weight

Children ≤ 20 kg body weight	
•	150 mg/kg acetylcysteine in 3 mL/kg 5% glucose over 60 minutes
•	Followed by 50 mg/kg in 7 mL/kg 5% glucose over 4 hours
•	Followed by 50 mg/kg in 7 mL/kg 5% glucose over 8 hours
•	Followed by 50 mg/kg in 7 mL/kg 5% glucose over 8 hours
Children > 20 kg bodyweight	
•	150 mg/kg acetylcysteine in 100 mL 5% glucose over 60 minutes
•	Followed by 50 mg/kg in 250 mL 5% glucose over 4 hours
•	Followed by 50 mg/kg in 250 mL 5% glucose over 8 hours
•	Followed by 50 mg/kg in 250 mL 5% glucose over 8 hours

2. ANAPHYLACTOID REACTION

- N-Acetylcysteine can cause **anaphylactoid reactions** with vomiting, flushing, urticaria, angioedema, bronchospasm and rarely shock.
- Very rarely it can also cause respiratory depression, acute kidney injury and DIC.
- Reactions occur in around **20% of patients** and are more likely in **women, brittle asthmatics** and those **with low paracetamol levels**.
- Reactions can usually be controlled by **simply stopping the infusion**.
- If the reaction persists **10 mg IV chlorphenamine can be given** and **salbutamol nebulisers** added if bronchospasm is present. *Previous reactions are no longer considered a contraindication to the use of acetylcysteine.*

V. SALICYLATE OVERDOSE

- Salicylate poisoning is a relatively common cause of poisoning and effective early treatment can prevent organ damage and death.
- Poisoning can be classified as mild, moderate or severe depending upon the plasma salicylate level:
 - **Mild poisoning** = < 450 mg/L
 - **Moderate poisoning** = 450-700 mg/L
 - **Severe poisoning** = > 700 mg/L
- **CLINICAL FEATURES INCLUDE:**
 - Nausea and Vomiting
 - Tinnitus and Deafness
 - Sweating and Dehydration
 - Hyperventilation
 - Cutaneous flushing
 - Hyperpyrexia (particularly children)
 - Hypoglycaemia (particularly children)
 - Severe poisoning can cause convulsions, cerebral oedema, coma, renal failure, non-cardiogenic pulmonary oedema and cardiovascular instability.
- **INVESTIGATIONS SHOULD INCLUDE:**
 - Plasma salicylate level
 - Arterial blood gas: **Primary respiratory alkalosis** may occur, followed by concomitant **primary metabolic acidosis (RALMAC)**
 - Blood glucose level
 - Urea and electrolytes
 - Clotting profile
 - ECG
- **ECG ABNORMALITIES IN SALICYLATE OVERDOSE:**
 - *Widening of the QRS complex*
 - *AV Block*
 - *Ventricular Arrhythmias*
- **TREATMENT**
 - Involves stabilization of the ABCs as necessary, limiting absorption, enhancing elimination, correcting metabolic abnormalities, and providing supportive care.
 - No specific antidote is available for salicylates.
 - **Gastric lavage** and **activated charcoal** (50 g) are indicated if greater than 4.5 g has been ingested in the previous hour (or > 2 g in a child).
 - Activated charcoal both reduces absorption and increases elimination of salicylate.
 - Severe cases usually require **aggressive intravenous fluids** to correct dehydration and **1.26% sodium bicarbonate administration**, which increases elimination of the salicylate.
 - **The urine pH** should be maintained at greater than **7.5** and ideally should be between **8.0-8.5**.
 - There is, however, no longer any role for **forced alkaline diuresis**.
 - Life-threatening cases will require intensive care admission, intubation and ventilation and possibly **haemodialysis**.

VI. DIGOXIN TOXICITY

• Overview

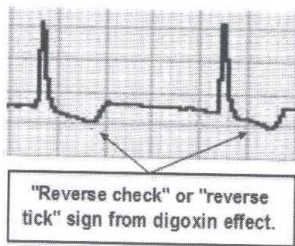
- Digoxin is a cardiac glycoside that primarily works by inhibiting the **Na⁺/K⁺ ATPase** in the myocardium.
- This results in a **slowing of the ventricular response** and a **positively inotropic effect**.
- Digoxin has a long half-life and maintenance doses need to be given only once daily.
- It should be monitored to ensure that the correct dosage is being given and to ensure that factors that can provoke toxicity (e.g. **renal dysfunction and hypokalaemia**) are not developing.
- Regular monitoring of plasma digoxin concentrations during maintenance treatment is not necessary once steady state has been achieved unless problems are suspected.
- In atrial fibrillation, the best monitor of response to treatment is the ventricular rate.
- A target range of **1.0-1.5 nmol/L** should be aimed for but concentrations of **2 nmol/L** may be required. The plasma concentration alone cannot indicate toxicity reliably, but the likelihood of toxicity rises dramatically at levels above 2 nmol/L.
- **Hypokalaemia** predisposes to digoxin toxicity and can be managed by co-administration of a **potassium-sparing diuretic or potassium supplementation**.

• THE CLINICAL FEATURES OF DIGOXIN TOXICITY

- **General:** Weakness, Fatigue, General Malaise
- **Cardiac:** almost any arrhythmia or heart block
- **Neurological:** Headache, Facial Pain, Dizziness, Confusion, Delirium, Psychoses and Hallucinations
- **Gastrointestinal:** Anorexia, Nausea, Vomiting and Abdominal Pain.
- **Visual:** Blurred Vision, Xanthopsia (**yellow vision**)

ECG FEATURES OF DIGOXIN TOXICITY:

- PR interval Prolonged
- QRS Prolonged
- QT Shortened
- ST depression (**reverse tick/check sign**)
- T wave inversion
- Bradycardia
- AV Block or dissociation
- Ventricular ectopics



• ED MANAGEMENT OF DIGOXIN TOXICITY

- Stop the digoxin
- Involve the cardiology team and/or the Poisons Information Service
- Monitor pulse, blood pressure and cardiac rhythm
- Check urea and electrolytes, magnesium, and digoxin levels
- Correct serum potassium
- Correct serum magnesium
- Monitor ECG and treat arrhythmias as appropriate
- **Digibind** (digoxin-specific antibody Fab) is the antidote used for digoxin poisoning. The digoxin-specific antibody fragments have a higher affinity for digoxin than the receptor in the body.
- *It is expensive and rarely needed and its use should be reserved for cases of severe poisoning only.*

VII. LIDOCAINE TOXICITY

- There are several conditions that increase the potential for lidocaine toxicity:
- **Liver dysfunction** increases the risk of toxicity due to lidocaine being metabolized by the liver.
- **Low protein** increases the risk of toxicity because lidocaine is protein bound.
- **Acidosis** can also increase the risk of toxicity since acidosis increase the potential of lidocaine to dissociate from plasma proteins.
- **SIGNS OF SEVERE TOXICITY**
 - Sudden alteration in mental status, severe agitation or LOC +/- convulsions.
 - Cardiovascular collapse: bradycardia, conduction blocks, asystole and VT may occur.
 - Local anaesthetic (LA) toxicity may occur some time after an initial injection.
- **IMMEDIATE MANAGEMENT**
 - Stop injecting the LA and Call for help.
 - Maintain the airway and, if necessary, secure it with a tracheal tube.
 - Give 100% oxygen and ensure adequate lung ventilation.
 - Confirm or establish intravenous access.
 - Control seizures: a benzodiazepine, thiopental or Propofol in small incremental doses.
 - Assess cardiovascular status throughout.
 - Consider drawing blood for analysis, but do not delay definitive treatment to do this.
- **TREATMENT**
- **In circulatory arrest**
 - Start CPR using standard protocols (be prepared for longer CPR)
 - Manage arrhythmias using the same protocols
 - Give **intravenous Lipid Emulsion (Intralipid)**
 - Continue CPR throughout treatment with lipid emulsion
 - Recovery from LA-induced cardiac arrest may take >1 h
 - **Propofol is not a suitable substitute for lipid emulsion**
 - **Lidocaine should not be used as an anti-arrhythmic therapy**
- **Without circulatory arrest**
 - Use conventional therapies to treat hypotension, bradycardia, tachyarrhythmia
 - Consider intravenous lipid emulsion

VIII. LITHIUM OVERDOSE

- Lithium is commonly used as a maintenance treatment for bipolar affective disorder.
- Lithium poisoning occurs relatively frequently as it is used in a population that is at high-risk for overdose.
- Poisoning can also occur accidentally due to therapeutic overdosage due to its relatively **narrow therapeutic index**.
- The usual therapeutic range for lithium is **0.4-0.8 mmol/l** (but the range may vary between laboratories).
- Toxic effects are often seen at **levels > 1.5 mmol/l**.
- **There are three main categories of lithium poisoning:**
 - **Acute poisoning:**
 - Occurs in patients recently started on lithium.
 - The main symptoms are **gastrointestinal upset** (nausea, vomiting, abdominal pain and diarrhoea).
 - More severe cases progress to **tremor, ataxia and confusion**.
 - In severe cases there can be **convulsions, coma and renal failure**.
 - **Acute-on-chronic poisoning:**
 - Occurs in patients taking lithium regularly that increased their dose or taken too much.
 - Symptoms are similar to acute poisoning but serum levels can be difficult to interpret.
 - **Chronic poisoning:**
 - Occurs in patients on long-term lithium patients and is usually precipitated by the introduction of a new medication that has impaired renal function.
 - Symptoms are primarily neurological.
 - Mental status is often altered and can progress to coma and seizures if the diagnosis is unrecognized.
 - These patients are very difficult to treat.
- **INVESTIGATIONS**
 - U&E
 - Lithium level
 - Patients with lithium overdose should have their **urea and electrolyte levels** measured due to the risk of renal impairment and a **lithium level checked** (which is sent in a plain tube, not a lithium heparin tube).
- **MANAGEMENT**
 - Admit all with symptoms of toxicity or levels **> 2mmol/l**
 - All patients that have taken a lithium overdose should be observed for at least 24 hours.
 - **Gastric lavage** can be performed if the patient presents within 1 hour of overdose
 - **Whole bowel irrigation** may be considered for an overdose of slow-release tablets.
 - **Haemodialysis** is the treatment of choice for severe poisoning

Note: Activated charcoal should not be used as it does not absorb lithium.

IX. COCAINE OVERDOSE

- o *Please see Cocaine Associated Chest Pain Chapter 11-III*

X. OPIATE OVERDOSE

- Opioid poisoning is a relatively common Emergency Department presentation.
- Overdose can be secondary to recreational drug (e.g. heroin) or as a consequence of prescribed opioids (e.g. morphine sulfate tablets, dihydrocodeine).
- **The clinical features of opioid overdose include:**
 - o Reduced conscious level or coma, Reduced respiratory rate, Apnoea, **Pinpoint pupils**
 - o Hypotension, Cyanosis, Convulsions and Non-cardiogenic pulmonary oedema (with IV heroin usage).
 - o The main cause of death secondary to opioid overdose is **respiratory depression**, which usually occurs **within 1 hour of the overdose**.
 - o Vomiting is also common and aspiration can occur.
- **Naloxone 0.4-2mg intravenously** is the specific antidote for opioid overdose and will reverse respiratory depression and coma if given at sufficient dosage.
- It can also be given by intramuscular injection if the intravenous route is not feasible.
- As Naloxone has a shorter duration of action than most opioids, close monitoring and repeated injections are necessary according to the respiratory rate and depth of coma.
- The dose is generally repeated every **2-3 minutes to a maximum of 10 mg**.
- When repeated doses are needed naloxone may be given by a continuous infusion adjusted according to the vital signs.
- Initially the infusion rate can be set at 60% of the initial resuscitative IV dose per hour.
- In opioid addicts naloxone administration may precipitate a withdrawal syndrome with abdominal cramps, nausea and diarrhoea, but these usually settle within 2 hours.

XI. BENZODIAZEPINES OVERDOSE

- Benzodiazepines work by binding to the gamma sub-unit of the GABA-A receptor.
- Their binding causes a structural modification of the receptor that results in an increase in GABA-A receptor activity. The binding of benzodiazepines increases the frequency of channel opening events, which leads to an increase in chloride ion conductance and inhibition of the action potential. Benzodiazepine drugs rarely cause serious poisoning when taken alone but can potentiate the effects of other central nervous system depressants, such as alcohol and tricyclic antidepressants. Fatal overdose is very uncommon but can occur in the elderly or in patients with respiratory problems (COPD)
- **The clinical features of benzodiazepine overdose include:**
 - o Drowsiness, Dizziness and ataxia, Dysarthria,
 - o Coma, respiratory depression and hypotension (rare)
- **ED MANAGEMENT OF BENZODIAZEPINES OD**
 - o The mainstay of management of benzodiazepine overdose is **airway maintenance and ventilation**.
 - o **Flumazenil** is a specific benzodiazepine antagonist that can be useful in some cases.
 - o It acts rapidly (in less than 1 minute) but the effects are short-lived and last less than 1 hour. **The dose is 200 µg every 1-2 minutes** (max dose 3mg / hour).
 - o *Flumazenil should be avoided if the patient dependant on benzodiazepine or takes tricyclic antidepressants as it can precipitate a withdrawal syndrome in these patients.*
 - o *In these circumstances, it can cause seizures or cardiac arrest.*

XII. ECSTASY

- **3, 4-METHYLENEDIOXYMETHAMPHETAMINE (MDMA)**
 - Dose 30-150 mg/tablet
 - Toxicity - stimulation peripheral/central α and β adrenoceptors
 - Severe, fatal reactions following previously tolerated doses
 - Early deaths usually cardiac arrhythmias
 - Features - effects within 1 hour, Last 4-6 hours after 75-150 mg (up to 48hrs after 100-300 mg)
- **Clinical features**
- **Mild symptoms / signs**
 - Nausea, Vomiting, Sweating, anxiety
 - Dizziness, drowsiness, chest/abdo pain
 - Myalgia, insomnia, Mydriasis, tremor
 - Tachycardia, mild hypertension
 - Ataxia, nystagmus, Slight pyrexia
- **Severe signs**
 - Hypertonia
 - Hyperpyrexia ($>39^{\circ}\text{C}$)
 - Initial hypertension
 - Later hypotension
- **Life threatening**
 - Coma
 - Convulsions
 - Delirium
 - Rigidity
 - ARDS
 - Rhabdomyolysis
 - ARF
 - DIC
 - Hepatocellular necrosis
- **Management**
 - **Charcoal** if within 1 hour
 - **Observe** all cases for 6 hrs
 - **If Moderate**
 - BP, ECG, Temp for 12 hrs
 - **Diazepam** for anxiety , agitation
 - **B-blockers** for tachycardia
 - **Cooling & Dantrolene** if $T >39^{\circ}\text{C}$
 - **If severe**
 - Consider intubation
 - Beware metabolic acidosis
 - Monitor clotting / renal fxn

XIII. KORSAKOFF SYNDROME

- It is a type of dementia caused by **Thiamine (vitamin B1) deficiency**.
- It is most commonly seen in patients with a history of chronic alcoholism.
- **Korsakoff syndrome (K.S.)** is characterised by the presence of *Anterograde amnesia, Patchy retrograde amnesia and Confabulation*
- Many patients also have evidence of Aphasia, Apraxia, Agnosia or a Deficit of executive functioning.
- K.S. frequently co-exists with **Wernicke's encephalopathy**, which is characterised by the triad of *Ophthalmoplegia, Altered mental state and Gait disturbance (ataxia)*
- When these co-exist, it is referred to as the Wernicke-Korsakoff syndrome (WKS).

HAZARDOUS DRINKING

- This is defined as drinking more than twice the recommended daily limit – i.e. **more than 8 units for a man and 6 units for a woman**. This group can benefit from a brief intervention involving advice and information about reducing alcohol intake.

DEPENDENT DRINKING

- This is defined as drinking more than twice the recommended daily limit every day, or demonstration of other signs of dependence. This group do not benefit from brief intervention and need more complex management from specialist alcohol workers.
- You must ask the patient about signs of dependence:
 - *Do they feel a compulsion to drink?*
 - *Do they show signs of tolerance – i.e. can they have a heavy intake without becoming intoxicated or feeling drunk?*
- **THE "CAGE" QUESTIONNAIRE**
 - *Have you ever tried to Cut down your drinking?*
 - *Do you ever get Angry when people talk to you about your drinking?*
 - *Do you ever feel Guilty about your drinking?*
 - *Do you ever take an 'Eye opener'?*

'Yes' to 3 out of 4 questions indicates likely dependence

RECOMMENDED SAFE LIMITS OF ALCOHOL

- ❖ *For a man: 21 units per week, with a maximum of 4 units in one day*
- ❖ *For a woman: 14 units per week, with a maximum of 3 units in one day*

MANAGEMENT

- Prevention and treatment of thiamine deficiency: **Thiamine 100 mg orally daily**
- Prevention and treatment of thiamine deficiency in severe alcoholics: **Thiamine 100 to 200 mg IV daily for 3 days then Thiamine 100 mg orally daily.**
- Treatment of Wernicke's encephalopathy
 - **Thiamine 500 mg IV infusion over 30 minutes**, 3 times daily for 3 days
 - Then **Thiamine 250 mg IV or IM**, daily for 3 to 5 days or until clinical improvement ceases.
- **Supportive care monitoring**
 - Optimise nutrition
 - Treat underlying cause, e.g. Chronic alcoholism with liver disease
 - Treat complications e.g. Heart failure

XIV. IRON POISONING

- A 3½ year-old boy ingested 50 mg/kg of elemental iron 2 hours ago. He vomited once. His parents brought him to the emergency department for assessment. An abdominal x-ray has been performed confirming the ingestion. He now looks well and has age-appropriate vital signs.
- The treating Doctor, stationed at a remote hospital, has called you for advice.

Q1. What is the risk assessment?

- An ingestion of 50mg/kg is expected to cause gastrointestinal symptoms, but not systemic toxicity.
- **Risk assessment according to dose is:**
 - **<20mg/kg:** asymptomatic
 - **20-60mg/kg:** GI symptoms only
 - **60-120mg/kg:** potential for systemic toxicity
 - **>120mg/kg:** potentially lethal
- Note that this is based on the amount of elemental iron ingested. This varies considerably between different types of iron tablets, depending on the type of ferrous or ferric salt.

Q2. By what mechanism(s) does iron toxicity occur?

- Iron has local gastrointestinal effects followed by systemic effects (that do not occur without preceding GI toxicity following iron ingestion)
- **Local effects:**
 - **Corrosive injury** to the gastrointestinal mucosa resulting in vomiting, diarrhoea, hematemesis, melena and fluid losses that may result in hypovolemia.
- **Systemic effects:**
 - Although the exact mechanisms are uncertain, iron acts as a cellular toxin targeting the cardiovascular system and the liver, with secondary CNS effects, metabolic acidosis due to hyperlactemia and free proton production from the hydration of free ferric ions, and coagulopathy.

Q3. What are the toxicokinetics of iron poisoning?

- In overdose the finely tuned mechanisms that normally regulate gastrointestinal absorption of iron are overwhelmed and bioavailability is greatly increased.
- Once iron is absorbed into the systemic circulation iron is gradually moved intracellularly over 6 to 12 hours.
- Elimination is minimal.

Q4. What is the clinical course in severe iron toxicity?

- Iron poisoning classically follows **5 stages**, although the stages usually overlap, reflecting the two important phases of toxicity: Gastrointestinal and Systemic.
- **CLASSIC STAGES AND TIME COURSE OF IRON TOXICITY:**
 - **Stage 1 (Gastrointestinal): 0-6 hours:** Vomiting, diarrhoea, hematemesis, melena, abdominal pain. Significant fluid losses may lead to **hypovolemic shock**.
 - **Stage 2 (Latent): 6-12 hours:** Gastrointestinal symptoms wane and the patient appears to be getting better. During this time iron shifts intracellularly from the circulation.

- **Stage 3 (Metabolic/cardiovascular): 12-48 hours:** Cellular toxicity becomes manifest as vasodilative shock and third-spacing, **high anion gap metabolic acidosis** (HAGMA) and **hepatorenal failure**.
- **Stage 4 (Hepatic): 2-5 days:** acute hepatic failure, although rare mortality is high
- **Stage 5 (Delay): 2-6 weeks:** chronic sequelae occur in survivors; cirrhosis and gastrointestinal scarring and strictures.

Q5. What investigations are useful in iron poisoning?

- In addition to the usual screening tests in suspected tox cases (Glucose, Pregnancy test, ECG, paracetamol level, U&E, LFT, Coagulation) the following specific tests can be useful:
 - **Serum iron concentration**
 - Peak levels occur 4-6 hours following iron ingestion
 - After 6 hours, iron levels fall due to intracellular shift
 - Levels do not clearly correlate with clinical toxicity, but $> 90 \text{ micromol/l}$ (500 mcg/dl) is generally considered predictive of systemic toxicity (equivalent to $>60 \text{ mg/kg}$)
 - **Blood gas**
 - The presence of **HAGMA** is a useful marker of systemic toxicity
 - In the absence of iron levels a serum bicarbonate level can be used as a surrogate marker
 - **Abdominal x-ray:** can be used to confirm ingestion
- *For serum iron measurement, samples should be drawn **at least 4 hours post-ingestion**, to allow levels to reach steady state; however, levels drawn **more than 6 hours after ingestion** may underestimate toxicity because of ferritin binding and redistribution of iron.*

Q6. Describe the indications, administration and potential adverse effects of the antidote that can be used in iron toxicity.

- **Desferrioxamine** chelation therapy is an option for severe iron toxicity
- **INDICATIONS:**
 - *Level $>90 \text{ micromol/L}$ at 4-6 hours post-ingestion*
 - *Evidence of systemic toxicity*
 - *Shock*
 - *Metabolic acidosis*
 - *Altered mental status*
- **ADMINISTRATION:**
 - Initial infusion rate of **15 mg/kg/h** , reduced if hypotension occurs, may be titrated up to **40 mg/kg/h in severe toxicity**
 - Cardiac monitoring is mandatory
 - Desferrioxamine is made as a 5 mg/ml solution by reconstituting 500 mg in 5 ml sterile water then diluting up to 100 ml with normal saline or 5% glucose.
- **ADVERSE EFFECTS:**
 - *Hypersensitivity*
 - *Hypotension (with rapid or high-dose IV administration)*
 - *ARDS (with infusions $>24 \text{ h}$)*
 - *Toxic retinopathy*
 - *Yersinia sepsis* (the ferrioxamine complex is a siderophore that promotes growth)

- The infusion can be stopped when the patient is clinically stable and the serum iron level is <60 micromol/L. **Ferrioxamine** is excreted unchanged in the urine, which classically turns a *vin rose* colour.

Q7. What is the most important priority in the early management of severe iron toxicity?

- **Fluid resuscitation**
- Resuscitate with boluses of **10-20 mL/kg crystalloid** to prevent shock from gastrointestinal losses, vasodilation and third spacing.

Q8. What options for decontamination are there for iron toxicity and are they indicated in this case?

- Decontamination is not indicated in this case systemic toxicity is not expected based on risk assessment
- Iron – like other metals – **does not bind to activated charcoal** but **whole bowel irrigation** can be used for abdominal x-ray confirmed ingestions of >60 mg/kg.
- In potentially lethal ingestions (e.g. >120 mg/kg) **surgical or endoscopic removal of iron are options.**

Q9. Does this child need to be retrieved to a tertiary center?

- No
- Ingestions of **>40 mg/kg in children should be assessed at hospital.** Then:
 - Those asymptomatic at 6 hours with a negative abdominal x-ray can be discharged home.
 - Those with symptoms are admitted to hospital and may require **IV fluids.**
 - Patients with the potential for systemic toxicity may be best managed at larger hospitals where iron levels can be measured and iron chelation therapy administered if needed.
 - In hospitals where serum iron levels are unavailable a **serum bicarbonate** can be used as a surrogate marker of systemic toxicity.

TOXIC ALCOHOL: METHANOL AND ETHYLENE GLYCOL

- Metabolites cause toxic effects
- **Features:**
 - Similar to alcohol (without the smell)
 - **Methanol:** Blindness due to retinal injury, latent
 - **Ethylene Glycol:** renal failure with rapid progression
 - High Anion gap for both
- **Treatment:**
 - Antidote: **Ethanol, Fomepizole**
 - **Dialysis**

CALCIUM CHANNEL AND BETA BLOCKERS TOXICITY

- Both cause AV blockade
- Results in myocardial depression and bradycardia
- **Treatment:**
 - **CCB:** Atropine, Calcium, High-dose Insulin-Dextrose
 - **Beta-Blockers:** Atropine, IV Glucagon, High-dose Insulin-Dextrose

XV. DRUG INDUCED HYPERTHERMIA

INTRODUCTION

- This review considers the three main forms of drug related hyperthermia:
 - Malignant hyperthermia,
 - Neuroleptic malignant syndrome
 - Serotonin syndrome.

LABORATORY INVESTIGATIONS TO BE CONSIDERED INCLUDE:

- FBC (leucocytosis is common in NMS), U&E, Lactate
- CK (elevated in NMS and for detection of rhabdomyolysis as a complication)
- Clotting, Toxicology screen
- CT head and lumbar puncture to be considered if central nervous system aetiologies are suspected

GENERAL MANAGEMENT OF DRUG-INDUCED HYPERTHERMIA IN THE ED

- **Discontinue** causative agent.
- Ensure **adequate ABC**: Airway protection, Breathing and Circulation
- Consider administration of **activated charcoal** if **within 1 hour** of ingestion and patient able to protect own airway.
- **Control hyperthermia** by reducing excessive muscle activity from agitation, seizures or shivering with the use of **benzodiazepines for sedation**.
- In severe cases (temperature $>41.1^{\circ}\text{C}$) the patient is likely to **require intubation and paralysis**.
- **External cooling measures** e.g. cooling blankets, ice packs, ice water submersion, cool water mist and fans.
- **Volume replacement** as indicated.
- Patients with moderate to severe symptoms will require treatment in a **HDU or intensive care setting**.
- Treat complications: Respiratory dysfunction, seizures, vomiting and diarrhoea, rhabdomyolysis, acute kidney injury, hepatic injury, DIC, multi-organ failure and death

1. MALIGNANT HYPERTHERMIA

- It is a life-threatening complication of anaesthesia.
- **Pathophysiology**
 - Following exposure to a trigger, excessive jaw rigidity, excessive carbon dioxide production, hyperthermia and tachycardia develop.
 - As ATP is used up, lactate production increases with a resulting **metabolic acidosis**.
 - Muscle breakdown leads to potentially fatal **hyperkalemia**.
 - Triggering agents include **inhalational anaesthesia (Halothane, Enflurane, Desflurane, Sevoflurane, Isoflurane)** and the **depolarising agent suxamethonium**.
 - Previous exposure to known triggering agents does not rule out the disease.
 - Excessive exercise in warm conditions can also trigger a reaction in those who are susceptible. During a reaction, there are significant increases in noradrenaline and increased survival has been demonstrated with alpha-blockade in animal models.
 - Elevated levels of serotonin appear during malignant hyperthermia and serotonergic drug have exaggerated responses in susceptible swine but serotonin antagonists have not been shown to be effective.

TREATMENT OF MALIGNANT HYPERTHERMIA

- General management: as above
- Specific management:
 - Dantrolene 1mg/kg IV every 5 minutes to a maximum dose of 10mg/kg.
 - Treatment of hyperkalaemia accordingly.
- Patient who have thought to have had an episode of malignant hyperthermia need to be referred to a malignant hyperthermia centre for investigation and genetic counselling.

2. NEUROLEPTIC MALIGNANT SYNDROME

- NMS is a rare idiosyncratic reaction occurring in patients that are taking neuroleptic drugs or after sudden withdrawal of dopamine agonists.
- Pathophysiology
 - Neuroleptic syndrome can occur at any time; even after years of therapy but is more likely to develop **within 10 days**.
 - Drug levels are often found to be therapeutic in neuroleptic malignant syndrome.
 - Butyrophenones and phenothiazines are most commonly implicated though at least 25 agents have been identified as triggers.
 - Some patients will develop neuroleptic malignant syndrome with any dopamine agonist, some will develop neuroleptic malignant syndrome with specific dopamine agonists whilst others can be treated with the same drug without any ill effect.

DRUGS CAUSING NMS

Atypical Antipsychotics	Typical Antipsychotics	Antiemetics
Chlorpromazine	Aripiprazole	Domperidone
Fluphenazine	Clozapine	Droperidol
Haloperidol	Olanzapine	Metoclopramide
Perphenazine	Paliperidone	Prochlorperazine
Thioridazine	Quetiapine	Promethazine
Thiothixene	Risperidone	

Clinical features

- Hyperthermia, Altered mental status, Skeletal muscle rigidity
- Autonomic dysfunction. A temperature of 38°C or above is a key diagnostic feature.
- Autonomic dysfunction manifests as tachycardia, hypotension or hypertension and diaphoresis. Mental status changes often precede muscle rigidity.
- It is often difficult to differentiate between neuroleptic malignant syndrome and serotonin syndrome in patients presenting with muscular rigidity, hyperthermia and autonomic instability. *Patients with serotonin syndrome present **within 24 hours of starting the medication**, whilst those with neuroleptic malignant syndrome present at anytime with peak symptoms not occurring for days.*

ED MANAGEMENT OF NMS

- General management: as above
- Specific management:
 - Bromocriptine (a dopamine agonist) 2.5-10 mg 6 hourly
 - Amantadine 100 mg orally has been used as an alternative to bromocriptine
 - Coagulopathy should be treated with FFP and platelets
 - Dantrolene 1-2.5 mg/kg up to a maximum of 10 mg/kg/day. This is the treatment for malignant hyperthermia but its use has been described in NMS

3. SEROTONIN SYNDROME

- Serotonin syndrome is a predictable consequence of excess serotonergic agonism of central nervous system receptors and peripheral serotonergic receptors.
- It is not an idiopathic drug reaction.
- Most cases occur with a therapeutic concentration, not overdoses.
- The commonest drugs that precipitate serotonin syndrome are **Venlafaxine, Fluoxetine, Citalopram, Pethidine and Tramadol**.
 - **Ondansatron** blocks serotonin post synaptic receptors and can not induce this syndrome.
- **Clinical features**
 - In moderate intoxication, a core temperature of 40°C is not uncommon.
 - Physical examination includes mydriasis, hyperactive bowel sounds, diaphoresis with normal skin colour. Clonus (inducible, spontaneous and ocular) is the most important finding in establishing the diagnosis.
 - Hyperthermia and hypertonicity occur in life threatening cases.
 - Signs of excess serotonin range from tremor and diarrhoea in mild cases to delirium, neuromuscular rigidity and hyperthermia in life-threatening cases.
 - Serotonin syndrome can result from therapeutic drug use, self-poisoning or inadvertent interactions between drugs.
 - The increase in dose of a causative agent or the addition of a drug with pro-serotonergic effects may provoke a dramatic clinical deterioration.
- **ED MANAGEMENT OF SEROTONIN SYNDROME**
 - **General management:** as above
 - **Specific management:**
 - **Antidote: Cyproheptadine 12 mg orally or via NG tube**
 - Patients with serotonin syndrome with severe hypertension and tachycardia should be treated with short acting cardiovascular agents such as **Esmolol or Nitroprusside**.
 - Longer acting agents such as propranolol should be avoided due to the autonomic instability in this group of patients.
 - Other agents such as olanzapine, chlorpromazine, bromocriptine or dantrolene are not recommended for use in the treatment of serotonin syndrome

4. ANTICHOLINERGIC SYNDROME

- *The combination of increased muscle activity causing increased heat production and the impaired ability to sweat leads to hyperthermia.*
- Anticholinergic agents are associated with hyperthermia at both therapeutic and toxic doses. Symptoms arise as result of the blockade of both the central and peripheral muscarinic acetylcholine receptors.
- **Symptoms resulting from central muscarinic receptor blockade:**
 - Altered mental status, confusion, restlessness, seizures, coma
- **Symptoms resulting from peripheral muscarinic receptor blockade:**
 - Impaired sweat gland function, Dry mouth, Dry axillae, Mydriasis, Tachycardia
 - Flushing, Urinary retention
- The onset of anticholinergic symptoms depends upon the drug but usually occurs **within a couple of hours of ingestion**.

- **Agents:** Antipsychotics, TCAs, Atropine, Antihistamines and Amphetamines
- **ED Management OF ANTICHOLINERGIC SYNDROME**
 - **Physostigmine 0.5-2 mg over 5 minutes** with continuous cardiac monitoring.
 - Most patients with anticholinergic syndrome improve with **supportive care alone**.
 - Supportive and general measures as previously described including **benzodiazepines** for the management of agitation and seizures.
 - **Phenothiazines and butyrophenones** are themselves anticholinergic so their use should be avoided in anticholinergic toxicity.
 - **Sodium bicarbonate** should be used in the case of arrhythmias or prolonged QRS intervals related to the anticholinergic poisoning.

5. CHOLINERGIC SYNDROME

- **NMJ:** weakness, Flaccid paralysis
- **Parasympathetic: DUMBELS:** Diarrhoea, Urination, Miosis, Bronchospasm, Emesis, Lacrimation, Salivation
- **Sympathetic:** Mydriasis, sweating, increased HR and BP
- **CNS:** agitation, confusion, Fits
- **Agents:** Organophosphates, Donepezil, Nerve agents, Neostigmine and Physostigmine
- **Treatment:**
 - Personal Protective Equipment
 - Supportive: Secretion Management
 - **Atropine 2-5mg IV every 5 min till sign of atropinisation appear**
 - **Pralidoxime 1-2g IV infusion over 15-20min**

6. SYMPATHOMIMETIC SYNDROME

- Sympathomimetic agents can cause life-threatening hyperthermia although the exact mechanism is unknown.
- Sympathomimetics cause a central increase in the concentrations of norepinephrine, dopamine and serotonin whilst peripherally causing a vasoconstriction, increased muscle activity and impaired behavioural responses. The degree of hyperthermia is not directly related to drug, mode of administration or duration. The agents which are most commonly associated with hyperthermia are **Amphetamine, Metamphetamine, MDMA and Cocaine**. Symptoms of sympathomimetic syndrome include agitation, altered mental status, hallucinations, coma, and seizures.
- Hyperthermia caused by sympathomimetics can also exacerbate these symptoms.
- **MANAGEMENT IN THE ED**
 - General measures and supportive treatment as described previously.
 - There is **no specific antidote** to treat the hyperthermia in sympathomimetic poisoning.
 - Treatment should aim for control of hyperthermia by reducing excessive muscle activity and supportive care to normalise vital signs.
 - Treatment might also be required for associated features such as hyponatraemia, hypertension and myocardial ischaemia.
 - Sympathomimetics such as cocaine and MDMA might also cause serotonin toxicity.
 - If there are features of serotonin toxicity as suggested by the Hunter diagnostic criteria, then consider treatment with **cyproheptadine** alongside supportive measures.

XVI. ACTIVATED CHARCOAL

- Multiple-dose activated charcoal involves the administration of more than 2 doses of oral activated charcoal to enhance elimination of drugs ingested in acute poisoning.
- The rationale is that charcoal interrupts the enteroenteric, enterogastric, and enterohepatic circulation of absorbed drugs, whereas unabsorbed drugs will be adsorbed to activated charcoal.
- The charcoal is prepared from vegetable matter such as peat, wood, coal, or coconut shell.
- It is then activated by high heat in oxidizing gas, such as steam or carbon dioxide, that increases its surface area to at least 900 m²/g to meet industry standards.
- **The optimal dose of charcoal is unknown.** However, the adult dose ranges from **50 to 100 g per dose, administered at a rate no less than 12.5 g/h or its equivalent.**
- Lower doses of **10-25 g are used in children (1g/Kg).** When drug-induced vomiting is anticipated (for example, with a theophylline overdose), an intravenous antiemetic is recommended.
- It does not work for poisonings by:
 - *Cyanide/ Iron/Lithium/ Corrosive agents,*
 - *Organophosphates/ Inorganic salts (K⁺)*
 - *Alcohols, Glycoles (ethylene glycol)*
 - *Metals (mercury, arsenic)/ Fluoride*
- Core recommendations are summarized as follows:
 - Multiple-dose activated charcoal should only be considered if a patient has ingested a life-threatening amount of **carbamazepine, dapson, phenobarbital, quinine, or theophylline.**
 - There are insufficient data to support or exclude the use of activated charcoal for the elimination of amitriptyline, dextropropoxyphene, digoxin, digitoxin, disopyramide, nadolol, phenylbutazone, phenytoin, piroxicam, and sotalol.
 - **The use of multiple-dose charcoal for salicylate poisoning** is controversial. One animal study and 2 of 4 volunteer studies did not demonstrate improved clearance, whereas 2 volunteer studies suggested improvement.
 - On the basis of existing experimental and clinical studies that show no efficacy, multidose activated charcoal is not recommended for the elimination of astemizole, chlorpropamide, doxepin, imipramine, meprobamate, methotrexate, sodium valproate, tobramycin, and vancomycin. The need for concurrent administration of cathartics such as sorbitol remains unproven and is not recommended. In particular, these should not be used in children because of possible fluid and electrolyte disturbances.
- **ABSOLUTE CONTRAINDICATIONS INCLUDE:**
 - *Unprotected airway*
 - *Intestinal obstruction*
 - *Gastrointestinal tract that is not intact*
 - *Decreased peristalsis/Ileus*
- **POTENTIAL COMPLICATIONS INCLUDE:**
 - *Transient constipation (especially in nonambulatory patients),*
 - *Bowel obstruction,*
 - *Regurgitation*
 - *Aspiration, with consequent pulmonary complications (including death)*

XVII. WHOLE BOWEL IRRIGATION

Introduction

- Gastrointestinal (GI) decontamination is a cornerstone in the general management of poisoned patients.
- The rationale behind GI decontamination is **to prevent absorption of ingested toxins** by either eliminating the toxin from the GI tract or binding the toxin within the GI tract.
- **INDICATIONS FOR WBI**
 - Ingestion of **large amounts of a toxin that is known to be poorly adsorbed by Activated Charcoal (AC)**.
 - Ingestion of massive amounts of drugs where **administration of adequate dosages of AC is impractical**
 - Ingestion of **sustained-release or enteric-coated drug preparations** (theophylline, calcium channel antagonists, aspirin, etc.)
 - **Removal of ingested packets of illicit drugs** (body packers, drug "mules")
 - Treatment of **suspected drug concretion** (i.e. continual rise in measurable toxin concentrations despite charcoal administration)
- **CONTRAINDICATIONS FOR WBI**
 - *Unprotected or compromised airway*
 - *Ileus/ Bowel obstruction or perforation*
 - *Uncontrollable vomiting*
 - *Severe GI hemorrhage*
- **POTENTIAL COMPLICATIONS**
 - *Nausea and vomiting*
 - *Abdominal cramping and bloating*
 - *Pulmonary aspiration*
- **WBI SOLUTION**
 - **Several polyethylene glycol electrolyte solutions (PEG-ES)** are available for use (Klean-prep, GoLyte, CoLyte).
 - These solutions are isotonic, osmotically balanced, and not absorbed into the body.
 - Originally designed for use as preoperative bowel cleansing preparations, PEG-ES solutions are routinely used for this indication as well as preparing the bowel for endoscopic or radiographic procedures.
 - Their safety is clearly demonstrated in clinical use without any appreciable changes in serum electrolytes or shifts in body fluids.
 - These solutions are also safe for use during pregnancy.
- **HOW TO PERFORM WBI**
 - Whole bowel irrigation is easily performed with unsophisticated equipment found in any emergency department.
 - However, expecting patients to drink adequate amounts of WBI fluid is unrealistic.
 - Therefore, it may be helpful to place a **nasogastric (NG) or small bore cilastin feeding tube** to administer the WBI solution.
 - The feeding tube is adequate for administering large quantities of WBI solution and is more comfortable for the patient than the typical stiff NG tube.
 - The WBI solution should be administered by gravity.
 - The infusion pump typically used to administer enteral feeding should not be used, as the typical maximal infusion rate (300 mL/hour) is inadequate.

- Antiemetics should be provided as needed.
- An agent with promotility effects (such as metoclopramide) is theoretically preferable.

Suggested Goal Rates for WBI Solution

9 months to 5 years	500 mL/hr
6-12 years	1000 mL/hr
13 years and older	1500-2000 mL/hr

- The recommended endpoint for WBI is clear rectal effluent.
- The co-administration of AC can complicate interpretation of this endpoint.
- When AC is present, PEG-ES should be administered until rectal output is "mostly" clear although some AC will likely be present in the output.
- This can typically be achieved after 8-10 liters (4-5 hours) of PEG-ES is administered.
- When toxins are amenable to monitoring by either radiographs (i.e. iron) or serial blood/serum concentrations, these techniques should be used to help guide the decision to stop or continue WBI. If collaborative evidence exists for persistence of drug in the GI tract (i.e. toxin seen on radiograph or rising drug concentrations), WBI irrigation should be continued until toxin is no longer visible or levels are clearly declining.

ANTIDOTES

01	Paracetamol	N-Acetylcysteine or Mucomyst/ Methionine
02	Warfarin	Prothrombin Complex Concentrate (PCC) or Vit K
03	Benzodiazepines	Flumazenil (Romazicon)
04	Beta-Blocking	Glucagon
05	Calcium Channel Blockers	Calcium; Anticholinergics
06	Dabigatran (Pradaxa)	Idarucizumab, Dialysis
07	Cyanide	Hydroxycobalamin, Na/Amyl Nitrite Dicobalt Edetate, Na Thiosulfate
08	Digoxin	Digoxin Immune Fab (Digibind)
09	Opioid (Heroin)	Naloxone (Narcan)
10	Iron	Deferoxamine
11	Heparin	Protamine Sulfate
12	Organophosphates	Atropine, Pralidoxime
13	Potassium	Insulin + Glucose, Kayexalate
14	Sodium channel blockers (TCAs), Salicylates	Sodium Bicarbonate
15	Ethylene glycol & Methanol	Ethanol / Fomepizole/ Dialysis / Thiamine
16	Local anesthetics	Intralipid/ Fat emulsion
17	Carbone Monoxide	Oxygen/Hyperbaric Oxygen
18	Heavy metals	Dimercaprol, Penicillamine, Na channel edetate
19	Paraquat	Charcoal, Fuller's earth
20	Antidepressants	Diazepam for convulsion, Bicarbonate for arrhythmia
21	Aspirin	Hemodialysis
22	Lithium	Gut decontamination, Hydration, Dialysis
23	Methaemoglobin	Methylene Blue

CHAPTER 29. EYE EMERGENCIES

I. ATRAUMATIC RED EYE

Definition

- The red eye is a term used to describe the reaction of the eye to exogenous or endogenous inflammation or infection. It encompasses inflammatory processes originating in the conjunctiva, episclera, sclera and anterior uveal tract.
- Non-traumatic subconjunctival haemorrhage is also commonly included in the causes of red eye although it is usually not part of an inflammatory or infective process.

1. CONJUNCTIVITIS

- Conjunctivitis is the commonest cause of red eye and can be bacterial, viral or allergic.

A. BACTERIAL CONJUNCTIVITIS

- Common causative organisms include:
 - **Strep. pneumoniae, staph. aureus and haemophilus influenzae.**
 - **Gonococcal and chlamydial** conjunctivitis are rarer but potentially far more serious and should be suspected in:
 - Neonates
 - Adults with urogenital symptoms i.e. urethral or vaginal discharge
 - Patients who fail to respond to initial treatment.
 - If either gonococcal or chlamydial conjunctivitis suspected, **an eye swab should be taken and the patient referred for an ophthalmology assessment.**

B. VIRAL CONJUNCTIVITIS

- Is commonly caused by infection with **adenovirus**, but other viruses (e.g. mumps, measles and herpes) may also be responsible.
- The infection is easily transmitted from eye to eye and between family members.
- Differentiating between viral and bacterial conjunctivitis, clinically, is unreliable and cannot be used to guide treatment.
- A more recent study has suggested that three indicators may prove useful to distinguish bacterial from viral conjunctivitis:
 - Early morning eyelid stickiness makes bacterial conjunctivitis more likely
 - Itching and a previous history of conjunctivitis both favour a viral aetiology
- Treatment of infective conjunctivitis with topical antibiotics is controversial.
- **Always prescribe topical antibiotics:**
 - Purulent / mucopurulent secretion and patient discomfort and ocular redness
 - Patients and staff in nursing homes, neonatal units, critical care units etc
 - Children going to nursery
 - Contact lens wearers
 - Patients with dry eyes or corneal epithelial disease
- **Usually prescribe topical antibiotics:**
 - Purulent / mucopurulent secretion and severe ocular redness
 - Patients with previously known external ocular disease
- **Delayed prescription or no antibiotic treatment:**
 - Patients who do not want immediate antibiotic treatment
 - Patients with moderate mucopurulent discharge and little or no discomfort
 - Co-operative and well informed patients

2. ALLERGIC CONJUNCTIVITIS

- Allergic conjunctivitis is typically a seasonal problem and commonly associated with other atopic diseases.
- Symptoms include itching and watering of the eyes, and findings on examination include bilateral generalised conjunctival injection, eyelid swelling and occasionally conjunctival oedema.
- The problem is managed by removing exposure to the pathogen, application of cool compresses to the eyes and in severe cases, an **oral or topical ocular antihistamine**.

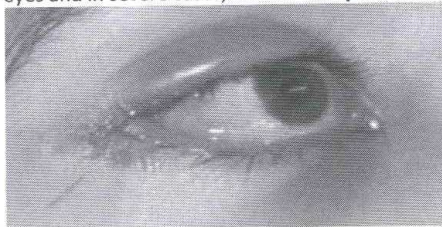


Figure 1.29.1. Oedema of the conjunctiva in allergic conjunctivitis

3. NON-TRAUMATIC SUBCONJUNCTIVAL HAEMORRHAGE

- This problem occurs when conjunctival or episcleral vessels bleed into the subconjunctival space. In spontaneous (non-traumatic) haemorrhage, the cause may be a **Valsalva manoeuvre** (e.g. coughing) or **trivial trauma**.
- Other systemic causes must be excluded and the patients **blood pressure** and **coagulation status** (if taking anticoagulant medication) need to be checked.
- Provided no other cause is found, the patient should be reassured and informed that the haemorrhage normally **takes 2-3 weeks to resolve completely**.



IMAGE A

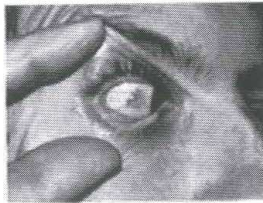


IMAGE B

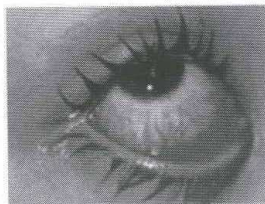


IMAGE C

Figure 1.29.2. A: Subconjunctival Haemorrhage B: Episcleritis C: conjunctivitis

4. KERATITIS AND KERATOCONJUNCTIVITIS

- Inflammation of the cornea, either alone (keratitis) or in combination with conjunctivitis (keraconjunctivitis), is distinguished by symptoms of pain, photophobia and reduction in visual acuity.
- Examination may show localised opacification of the cornea but more typically, fluorescein staining from **corneal ulceration** in a **punctuate**, rounded or branching (dendritic) pattern seen in **herpes simplex keratitis**.
- Bacterial keratitis is rare and is more common in contact lens wearers where staphylococcus and pseudomonas aeruginosa are the most frequent causative organisms.

- Primary HSV epithelial keratitis usually resolves spontaneously, however, treatment with antiviral medication does indeed shorten the course of the disease and may therefore reduce the long term complications of HSV.

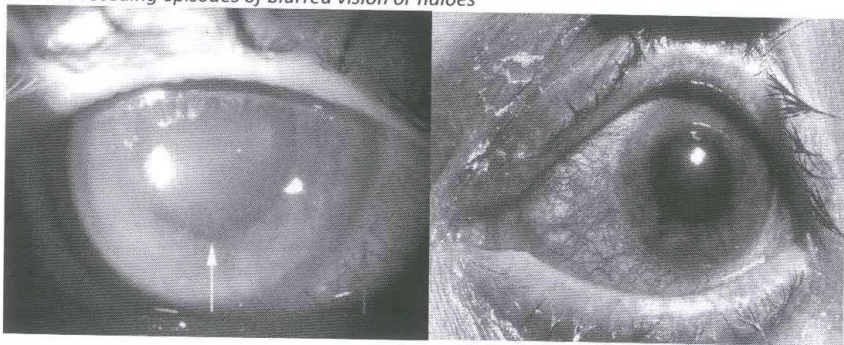


Figure 1.29.3. Herpes Simplex Keratitis

- The mainstay of therapy is antiviral treatment either in the form of topical therapy with **trifluridine 1% 8-9 times a day** or oral administration of **acyclovir or valacyclovir for 10 to 14 days**.
- **Topical corticosteroids are contraindicated** in the treatment of active HSV epithelial keratitis.
- All patients with keratitis, keratoconjunctivitis or corneal ulcer must be reviewed by an ophthalmologist for further investigation, treatment and follow-up.

5. ACUTE ANGLE CLOSURE GLAUCOMA (AACG)

- Acute closed-angle glaucoma is an ophthalmological emergency.
- It is caused by a blockage of fluid access to the trabecular meshwork by the iris bowing forwards and coming into contact with it at the entrance to Schlemm's canal. This results in a rise in intra-ocular pressure and a subsequent glaucomatous optic neuropathy.
- **CLINICAL FEATURES**
 - Severe eye pain
 - Loss of vision or decreased visual acuity
 - Congestion and circumcorneal erythema
 - Corneal oedema and cloudy
 - A fixed semi-dilated ovoid pupil
 - Nausea and vomiting
 - Preceding episodes of blurred vision or haloes



- **RISK FACTORS**

- Female gender (Female to male ratio 4:1)
- African or Asian ethnicity
- Hypermetropia (long-sightedness)
- Increasing age (anterior chamber becomes shallower)
- Family history of glaucoma
- Diabetes mellitus

- **THE ED MANAGEMENT OF CLOSED-ANGLE GLAUCOMA CAN INCLUDE:**

- **Topical 2% Pilocarpine drops** to both eyes **every 15 minutes**
- **Topical 0.5% Timolol drops**
- IV Morphine titrated to pain
- IV anti-emetic e.g. Metoclopramide
- IV Acetazolamide 500mg
- Urgent referral to on-call ophthalmologists
- Definitive treatment is a **laser iridotomy or iridectomy**.

6. EPISCLERITIS

- Localised, engorgement of the superficial episcleral plexus is the hallmark of this condition which may occur in isolation or together with uveitis or keratitis.
- Although normally benign, episcleritis may be associated with rheumatological and inflammatory diseases such as **rheumatoid arthritis, sarcoidosis and inflammatory bowel disease**.
- Patients describe irritation, rather than pain, in the eye with a mild watery discharge and no disturbance of vision.
- **MANAGEMENT:**
 - Patients should be reassured and advised that the condition is self-limiting.
 - **A topical NSAID agent** may ease discomfort.

7. SCLERITIS

- Scleritis is an inflammatory process involving the deep episcleral plexus and in 30-50% of cases is associated with an underlying rheumatological disorder.
- **Rheumatoid Arthritis** and **Wegener's Granulomatosis** are the most common connective tissue and vasculitic causes.
- Patients present with either localised or generalised bluish or violet discolouration and a deep dull aching pain in the eye.

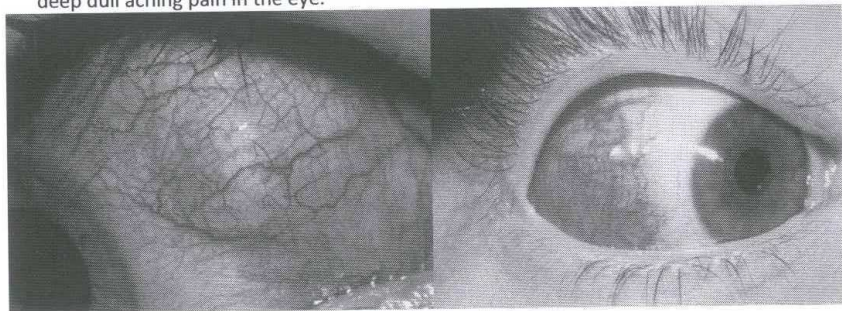


Figure 1.29.4. Scleritis and Episcleritis

- The pain is characteristically worse at night and may wake the patient from sleep.
- Vision may be affected and, as the extra-ocular muscles attach to sclera, the pain is worse on movements of the eye. The eye is tender to touch through the closed eyelid.
- *Differentiation between episcleritis and scleritis may be difficult. However, instillation of 2.5% phenylephrine drops into the affected eye results in blanching of the superficial episcleral plexus after 5 minutes. Therefore, persisting vascular engorgement indicates scleritis.*

- **MANAGEMENT:** urgent ophthalmological assessment.

8. ANTERIOR UVEITIS (IRITIS)

- Anterior uveitis is inflammation of the iris and typically presents with a painful, red eye with associated photophobia, lacrimation and decreased visual acuity. A hypopyon (pus in the anterior chamber) is sometimes seen.
- It is strongly associated in 70% of cases with the **HLA-B27 serotype**.
- Although sometimes occurring in eye disorders such as herpetic keratitis and following recent intraocular surgery, it is more commonly associated with diseases such as **Sarcoidosis, Ankylosing Spondylitis and Inflammatory Bowel Disease**.
- Anterior uveitis has numerous causes including:
 - Idiopathic (no cause found)
 - Trauma
 - Chronic joint disease e.g. spondyloarthropathies and juvenile chronic arthritis
 - Inflammatory bowel disease
 - Psoriasis
 - Sarcoidosis
 - Infectious e.g. Lyme disease, TB, leptospirosis, HSV and VZV
 - Malignancy e.g. Non-Hodgkins lymphoma, ocular melanoma and retinoblastoma

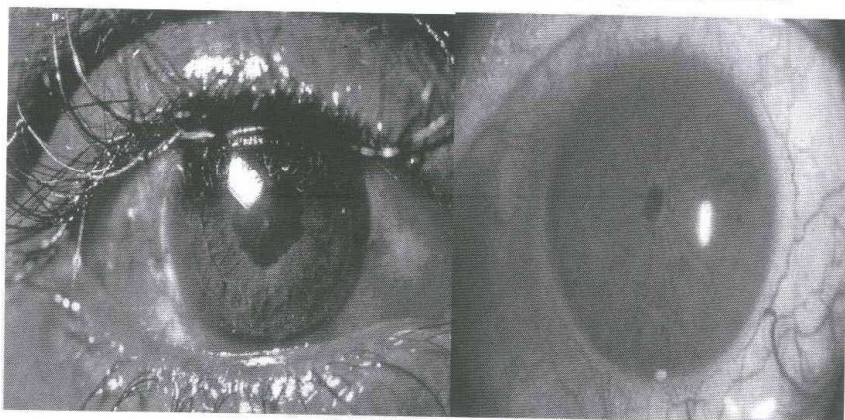


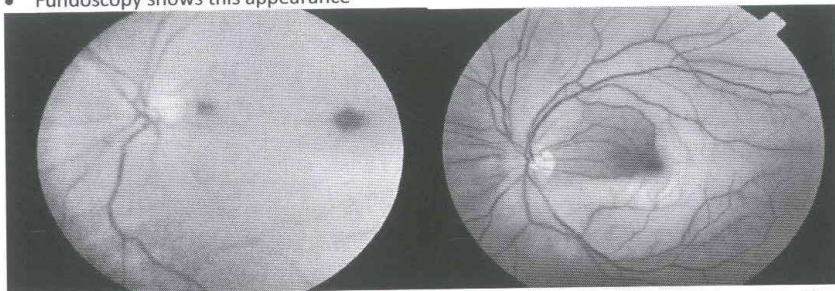
Figure 1.29.5. Anterior iritis

- A painful eye with perilimbal injection, photophobia and an irregular pupil are all indicative of anterior uveitis.
- The presence of keratic precipitates, inflammatory cells and flare confirm the diagnosis.
- **MANAGEMENT:** Urgent ophthalmology assessment.

II. VISUAL LOSS

1. CENTRAL RETINAL ARTERY OCCLUSION (CRAO)

- A 52 year-old man presents with sudden onset loss of vision in his right eye. He has no other symptoms. His past medical history is notable for hypertension, hyperlipidemia and angina. His medications include aspirin, atenolol, and atorvastatin. He can barely detect hand movements with his right eye and has a relative afferent pupillary defect.
- Fundoscopy shows this appearance



- Central retinal artery occlusion (CRAO) usually presents with **sudden, painless visual loss** in the affected eye.
- It can be caused by **emboli** from atheromatous carotid arteries, **thrombosis** secondary to arteriosclerosis or hypertension and vasospasm secondary to giant cell arteritis.
- **The pupil** on the affected side is *usually poorly reactive to light with a normal consensual light reaction*.

Q1. What is the diagnosis?

- **Central retinal artery occlusion (CRAO)**

- The diagnosis must be suspected in any case of sudden painless loss of vision, and is clinched by the appearance of the retina (see Q2).

Q2. What features on history and examination should be looked for?

• History

- Sudden and Painless loss of vision (seconds)
- Consider *underlying causes*: emboli, thrombosis, Giant Cell Arteritis (GCA), hypercoagulable, trauma, migraine, syphilis, sickle cell disease, Behcet's.

• Examination

- **Visual acuity** — markedly reduced e.g. <6/60
- **Marcus-Gunn pupils** (RAPD)
- **Red reflex** — abnormal and asymmetrical
- **Fundoscopy** — a pale retina with areas of cilioretinal sparing and a classic '**cherry red spot**' in the macula (may be subtle). Arteriolar and venular narrowing and box-car appearance.

'Cherry red spot' (Sparing of centre of the macula due to supply by underlying choroid)

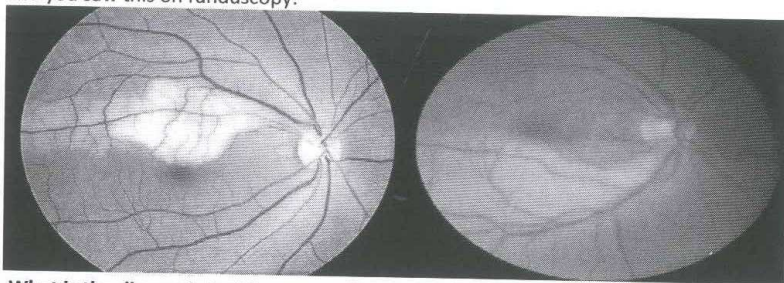
Q3. What is the investigation and management?

- CRAO, like chemical injuries to the eyes, is a true ophthalmological emergency.
- Urgent ophthalmology referral, and a physician to work up underlying causes.
- Urgent **ESR** and **CRP** to exclude **GCA**.
- TIA and vasculitis work up as per **amaurosis fugax**.

- Treatment is unproven but includes:
 - **Re-breathe CO₂**—to vasodilate the retinal artery.
 - **Timolol eye drops**—to reduce intraocular pressure.
 - **Acetazolamide 500 mg IV**—to reduce the production of aqueous humor and intraocular pressure.
 - **Massage the globe**—aiming to dislodge the embolus.
 - **Sublingual GTN**—to vasodilate the central retinal artery

Q4. How does an ophthalmic artery occlusion differ from this condition?

- Now, consider an alternative scenario in which the patient only had partial visual field loss and you saw this on funduscopy:

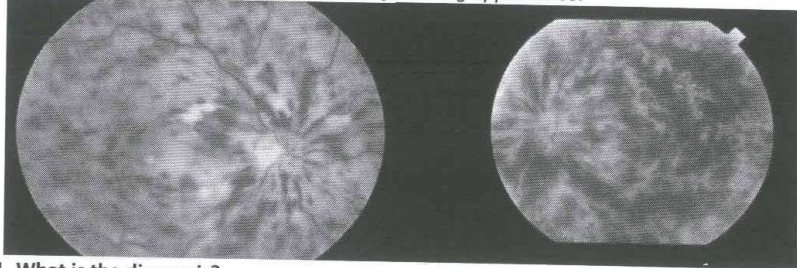


Q5. What is the diagnosis in this case, and how does it differ from the first?

- **Branch retinal artery occlusion (BRAO)**
- There is no cherry red spot and there is only partial visual field loss. A segment of retina is whitened along the distribution of a branch retinal artery. The treatment is the same.

2. CENTRAL RETINAL VEIN OCCLUSION (CRVO)

- A 56 year-old female presents with sudden onset loss of vision in her right eye. She has a past medical history of hypertension, hyperlipidemia and medication-controlled diabetes mellitus type 2. Her medications include aspirin, ramipril, atorvastatin and metformin. On examination she has 6/60 vision in her right eye.
- You perform funduscopy and observe the following appearance:



Q1. What is the diagnosis?

- **Central retinal vein occlusion**
 - Sudden painless loss of vision, in a patient with risk factors and a 'blood and thunder' retinal appearance (**Stormy sunset appearance**).

Q2. What are the predisposing factors and associated conditions?

- Glaucoma
- Old age

- Hypertension
- Diabetes mellitus
- Hypercoagulable state
- Atherosclerosis (vein is compressed by adjacent artery)
- Retrobulbar compressive lesions (e.g. Thyroid disease, orbital tumour)
- Vasculitis

Q3. What features on history and examination should be looked for?

• History

- *Sudden and painless loss of vision*
- *Assess for risk factors/ underlying causes (see Q2)*

• Examination

- **Visual acuity** — variable depending on severity and duration since onset
- **A Marcus-Gunn pupil** may be present if ischemic CRVO (relative afferent pupillary defect = RAPD)
- **Red reflex** — may be abnormal
- **Fundoscopy** — large areas of hemorrhage:
 - **Non-Ischemic CRVO**: dilated tortuous veins, retinal hemorrhages, cotton wool spots, retinal edema, disc swelling.
 - **Ischemic CRVO** (more severe): classic '**blood and thunder**' appearance (**Stormy sunset appearance**) from widespread hemorrhages that obscure most fundal details.

Q4. What is the management?

- **Refer to an ophthalmologist**: *photocoagulation* may be performed if there is neovascularisation.
- **Refer to a physician** for ongoing work-up and treatment of underlying causes
- **Screen for risk factors** (cardiovascular disease, diabetes, vasculitis, etc)
- **Consider low-dose aspirin** (unproven)

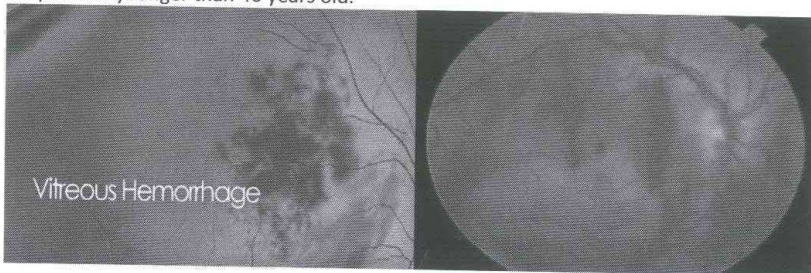
Q5. How does branch retinal vein occlusion differ from this condition?

- A **branch retinal vein occlusion** only affects a sector of the retina corresponding to the distribution of the affected branch. Visual loss is limited to a segment of the visual field.

3. VITREOUS HAEMORRHAGE AND RETINAL TEARS

- The vitreous body represents 80% of the eye and is 99% water and 1% hyaluronic acid/collagen. It fills the space between the lens and the retina. It is adherent to the retina in three places: anteriorly at the border of the retina, at the macula, and at the optic nerve. With increasing age, the vitreous may liquefy and the collagen fibres clump together, causing the vitreous to collapse. The pockets left by the collapsed vitreous after often seen as 'floaters'.
- Vitreous haemorrhage can occur due to rupture of abnormal blood vessels or due to stress on normal vessels.
 - **Abnormal vessels**: are typically the result of neovascularization due to ischaemia, most commonly from diabetic retinopathy or coagulopathies (e.g. sickle cell anaemia). The new vessels are fragile and more prone to rupture.
 - **Normal vessel rupture**: can occur when sufficient mechanical force is applied. If the vitreous detaches posteriorly it may pull on these vessels resulting in rupture. The force may also cause the retina to tear or detach.

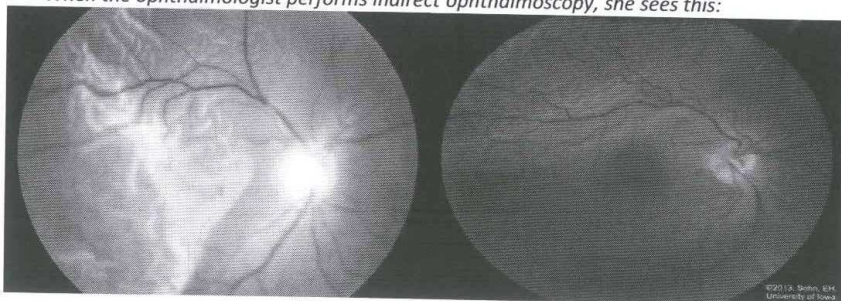
- Blunt or penetrating trauma can damage intact vessels and is the leading cause in patients younger than 40 years old.



- **Clinical features of vitreous haemorrhage**
 - Early or mild haemorrhage may present as floaters, cobwebs, haze, shadows, or a red hue. In large bleeds, visual acuity may be severely reduced.
 - Loss of red reflex.
 - Retina is difficult to visualise on fundoscopy.
- **ED MANAGEMENT OF VITREOUS HAEMORRHAGE**
 - Sit the patient head up to allow blood to collect inferiorly.
 - Refer to ophthalmology.
 - Urgent assessment is required to assess for an associated retinal tear.

4. RETINAL DETACHMENT

- A 50 year-old man presents with loss of vision. He describes a **curtain** coming down across his vision. It was preceded by '**flashes and floaters**'.
- When the ophthalmologist performs indirect ophthalmoscopy, she sees this:



Q1. What is the diagnosis?

- **Retinal detachment**
 - This is the separation of the sensory retina from the underlying pigmented retinal epithelium.
 - **Findings:**
 - **Ultrasound:** The detached retina is visible as a free floating echogenic membrane separated from the globe posteriorly. It moves with eye movement and is attached at the optic disc.
 - **Ophthalmoscopy:** The detached retina appears corrugated and partially opaque.
 - **Funduscopy:** the detached portion will appear out of focus.

Q2. What are the 3 types of mechanisms that can cause this condition?

- There are 3 types of retinal detachment:
 - **Rhegmatogenous:** the detached retina is elevated by underlying fluid that collects from the vitreous through a tear in the retina. This is the most common mechanism. It may be related to trauma, but is more common in men, those over age 45 years and those with myopia.
 - **Exudative:** fluid collects from retinal vessels. The causes may be neoplastic, inflammatory, congenital, or vascular in nature and include hypertension, pre-eclampsia, central retinal venous occlusion (CRVO), glomerulonephritis, papilledema, vasculitis, and choroidal tumours.
 - **Tractional:** the retina is pulled up by fibrocellular bands. This occurs in conditions such as proliferative diabetic retinopathy, sickle cell disease, retinopathy of prematurity (ROP), previous vitreous hemorrhage, trauma, and toxocariasis.

Q3. What are the features on history and examination?

- **History**
 - **Painless loss of vision** (central, peripheral or both)
 - Recent history of increased numbers of **flashes** (due to traction on the retina) and **floaters** (due to hemorrhage and debris in the vitreous).
 - Presence of a **dark shadow or curtain** moving over the visual field of the affected eye.
- **Examination**
 - **Visual acuity:** reduced if the macula is involved.
 - **Red reflex:** abnormal; a mobile detached retina may be visible.
 - **Visual fields:** reduced.
 - **Pupils:** a mild relative afferent pupillary defect (RAPD) may be present depending the size of the retinal detachment.
 - **Ophthalmoscopy:** The detached retina appears corrugated and partially opaque. On funduscopy the detached portion will appear out of focus. Other features that may be seen include: anterior vitreous pigmented cells, vitreous hemorrhage, and posterior vitreous detachment.

Q4. Describe investigation and management.

- **Investigation**
 - **Direct funduscopy** in the Emergency Department *cannot rule out retinal detachment*
 - **Ultrasound** is a useful investigation for diagnosing retinal detachment in the ED.
- **Management**
 - Urgent ophthalmologist opinion.
 - Minimise activity: bed rest with toilet privileges.
 - Treatment of underlying cause (especially if exudative).
 - Surgical options include laser photocoagulation, cryotherapy, pneumatic retinopexy, vitrectomy, and scleral buckle.
 - Close follow up is required.

Q6. What is a Retinal break?

- A **Retinal break** is a tear in the retinal membranes and may or may not lead to retinal detachment.

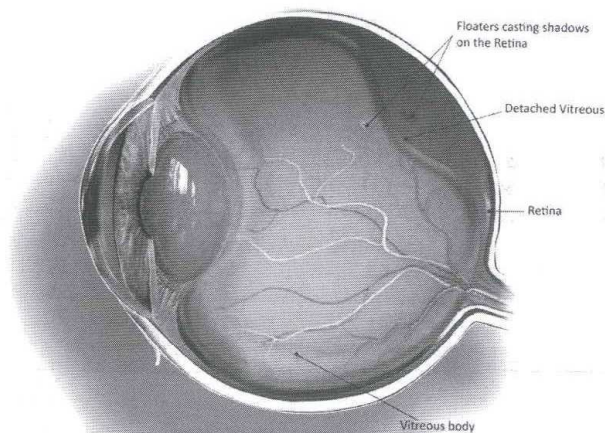
Q6. What is retinoschisis?

- **Retinoschisis** refers to the **splitting of the retina**, which has X-linked juvenile and age-related degenerative forms. It may be asymptomatic or lead to vision loss due to macular involvement and vitreous hemorrhage. It may be amenable to surgery.

5. POSTERIOR VITREOUS DETACHMENT (PVD)

- Occurs when the vitreous membrane separates from the retina.
- **Risk factors for posterior vitreous detachment include:**
 - Increasing Age
 - Diabetes Mellitus
 - Eye Trauma
 - Myopia
 - Recent Cataract Surgery
- **The main clinical features of posterior vitreous detachment are:**
 - Flashes of light (photopsia)
 - Increased numbers of floaters
 - A ring floaters to the temporal side of central vision
 - A feeling of heaviness in the eye
 - Weiss' ring (an irregular ring of translucent floating material in the vitreous)
- There is a small associated risk of retinal detachment in the 6-12 weeks following a posterior vitreous detachment.

POSTERIOR VITREOUS DETACHMENT



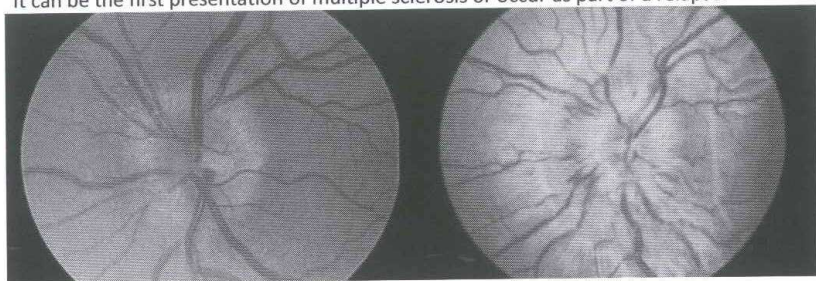
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Figure 1.29.6. Posterior Vitreous Detachment

- Retinal detachment can be distinguished from posterior vitreous detachment by the presence of:
 - A dense shadow in the periphery that spreads centrally
 - A 'curtain drawing across the eye'
 - Straight lines suddenly appearing curved (**positive Amsler grid test**)
 - Central visual loss and decreased visual acuity

6. OPTIC NEURITIS

- Optic neuritis is a demyelinating inflammation of the optic nerve.
- It affects the optic nerve peripheral to the **optic chiasm**.
- The commonest cause is **Multiple Sclerosis (MS)**.
- It usually presents with **sudden onset loss of vision**, which can be partial or complete, and painful eye movements.
- It can be the first presentation of multiple sclerosis or occur as part of a relapse.



• OTHER CAUSES INCLUDE:

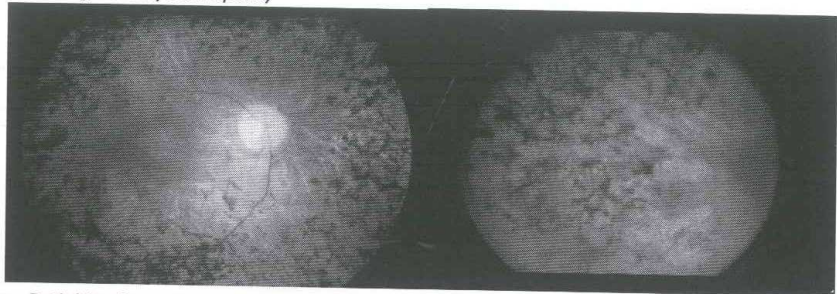
- *Infections e.g. Herpes zoster, Lyme disease*
- *Autoimmune disorders e.g. SLE, Neurosarcoidosis*
- *Poisoning e.g. Methanol*
- *Diabetes mellitus*
- *Vitamin B12 deficiency*
- Any sudden increase (i.e. over a 24-48 hour period) in symptoms of MS should be urgently assessed by a neurologist with expertise in the management of the condition.
- **Daily IV Methylprednisolone 500mg infusion/4hr X 5/7** treatment should be considered, and if initiated, should be done so at the earliest opportunity.

7. TEMPORAL ARTERITIS

- Temporal arteritis, also known as **Giant Cell Arteritis (GCA)**, is a type of chronic vasculitis characterized by granulomatous inflammation in the walls of medium and large arteries.
- It usually affects people over 50 years of age.
- **CLINICAL FEATURES INCLUDE:**
 - *Headache*
 - *Scalp tenderness*
 - *Jaw claudication*
 - *Amaurosis fugax or sudden blindness (typically unilateral)*.
- Some patients also present with systemic features such as fever, fatigue, anorexia, weight loss, and depression.
- It is associated with polymyalgia rheumatica (PMR) in 50% of cases (bilateral upper arm stiffness, aching, and tenderness; pelvic girdle pain).
- Visual loss occurs early in the course of disease and, once established, it rarely improves.
- Early treatment with **high-dose corticosteroids (40-60 mg prednisolone daily)** is imperative to prevent further visual loss and other ischaemic complications.
- An urgent referral for specialist evaluation (same day ophthalmology assessment for those with visual symptoms) and **temporal artery biopsy** should also be organised.

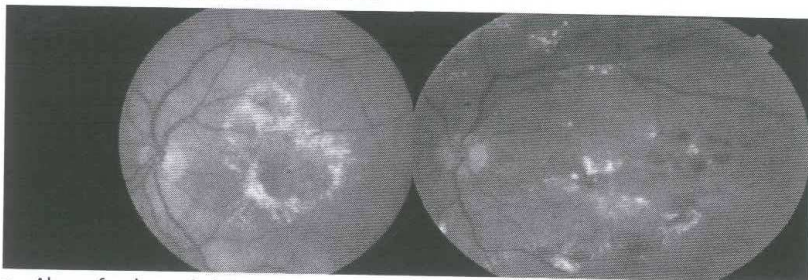
8. RETINITIS PIGMENTOSA

- A fundoscopic examination revealing bony spicule-shaped pigment deposits in the periphery with preservation of the macula is characteristic of retinitis pigmentosa.
- Retinitis pigmentosa is a group of inherited disorders characterized by:
 - *Night blindness (nyctalopia)*
 - *Loss of peripheral vision (tunnel vision)*
 - *Altered colour vision*
 - *Pigmentary retinopathy*



- Retinitis pigmentosa can be passed on by all forms of inheritance, but 50% of patients have no known affected relatives.
- There is often also an association with rare systemic disorders including:
 - *Laurence-Moon-Biedl-Bardet syndrome*
 - *Abetalipoproteinaemia*
 - *Refsum's disease*
 - *Kearns-Sayre syndrome*
 - *Usher's disease*
 - *Freidreich's ataxia*
- These patients should be referred on for genetic counseling and an ophthalmology assessment.

9. DIABETIC MACULOPATHY



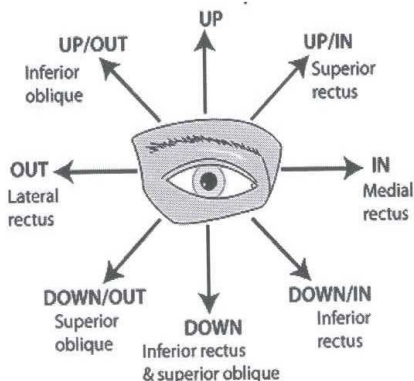
- Above fundoscopic images are consistent with that of diabetic maculopathy and patient should be referred for an ophthalmology opinion with 4 weeks.
- The following are the recommended referral criteria for diabetic retinopathy:
 - **Referral for an opinion within 4 weeks if:**
 - There is an unexplained drop in visual acuity

- There are hard exudates within 1 disc diameter of the fovea
- Macular oedema is present
- There are unexplained retinal findings
- Pre-proliferative or severe retinopathy is present
- **Referral to ophthalmology specialist within 1 week if:**
 - There is new vessel formation
 - There is evidence of pre-retinal and/or vitreous haemorrhage
 - Rubeosis iridis is present
- **Emergency referral to ophthalmology specialist on the same day if:**
 - There is sudden loss of vision
 - There is evidence of retinal detachment

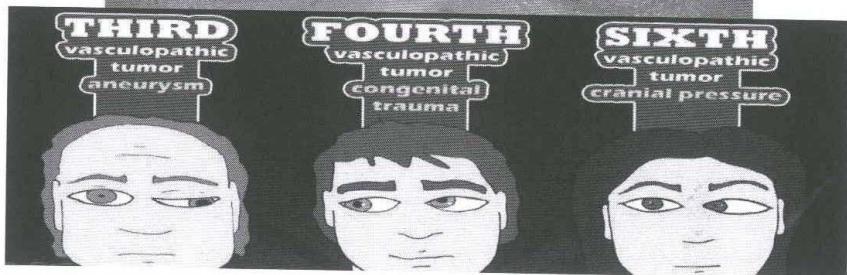
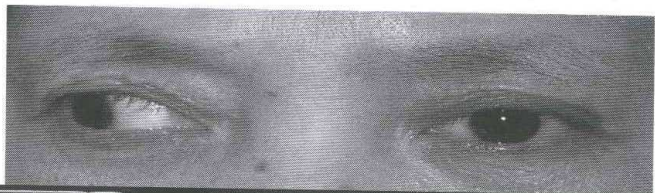
10. OCULAR NERVE PALSY

Nerve	Presentation	Causes
CN 3	<ol style="list-style-type: none"> 1. Failure of adduction, elevation and depression of the eye. The eye rests in the "down and out" position 2. Ptosis 3. +/- Pupil involvement (dilatation) 	<ul style="list-style-type: none"> • Microvascular disease: DM, HTN • Aneurysm of PCA • Tumour • Trauma • Infection: Herpeszoster
CN 4	<ol style="list-style-type: none"> 1. Failure of depression when the eye is in the adducted position (i.e. inability to look down towards the nose) 2. May manifest as vertical diplopia when reading/going down stairs 3. Patient classically have a compensatory head tilt 	<ul style="list-style-type: none"> • Trauma • Vascular disease : DM, HTN • Demyelinating disorders: Multiple sclerosis • Idiopathic • Congenital
CN 6	<ol style="list-style-type: none"> 1. Failure of abduction 2. Manifests as horizontal diplopia, that is worse when looking towards affected side 	<ul style="list-style-type: none"> • Trauma • Vascular disease : DM, HTN • Idiopathic • Less common: <ul style="list-style-type: none"> ○ Raised ICP, Tumour, Aneurysm, ○ Thrombosis of cavernous sinus, ○ MS, Post-viral syndrome in children

- *The superior rectus moves the eye up and in.*
- *The inferior oblique pulls the eye up and out.*
- *The inferior rectus pulls the eye down and in.*
- *The superior oblique pulls the eye down and out.*
- *The lateral rectus is responsible for moving the eye out and*
- *The medial rectus is responsible for moving the eye in.*



- Ocular muscles innervation: LR6 (SO4) Rest 3
- The features of a 3rd nerve palsy are:
 - Inability to move the eye superiorly, inferiorly and medially
 - Resting eye position '**down and out**'
 - Ptosis
 - Pupil fixed and dilated (mydriasis)
- The eye position at rest is '**down and out**' due to preservation of the superior oblique (moving the eye downwards) and lateral rectus (moving the eye outwards).
- **Compression from a posterior communicating artery aneurysm** is the commonest cause of a painful 3rd nerve (oculomotor nerve) palsy, making it the most likely diagnosis.
- These patients will require an urgent neurosurgical referral for angiography and/or surgical intervention.



1. NON-TRAUMATIC TRANSIENT VISUAL LOSS

- **COMMON CAUSES OF INCLUDE:**
 - Amaurosis fugax (usually minutes) — usually embolic or thrombotic; can occur secondary to hypoperfusion states, hyperviscosity or vasospasm.
 - Migraine (can be without headache)
 - One eye closed!

2. PAINLESS ACUTE PERSISTENT LOSS OF VISION:

- Central retinal artery occlusion (CRAO)
- Central retinal vein occlusion (CRVO)
- Retinal detachment or hemorrhage
- Vitreous hemorrhage
- Optic or retrobulbar neuritis
- Internal carotid artery occlusion

3. PAINFUL ACUTE LOSS OF VISION:

- Acute glaucoma
- Uveitis
- Endophthalmitis
- Keratoconus (vision can deteriorate rapidly and is associated with photophobia)

III. CORNEAL INJURIES

1. CORNEAL ABRASIONS

- Corneal abrasions result from scratching, cutting or abrading the protective epithelium of the cornea.
- After a minor abrasion, healthy cells quickly fill the defect and prevent infection or irregularity in refraction.
- Deeper penetration of the cornea results in the healing process taking longer from 24 to 72 hours.
- Patients with corneal abrasions present with an acute painful eye, commonly with a history of trauma. Other symptoms include:
 - Foreign body sensation
 - Blurred vision
 - Photophobia
 - Ophthalmoplegia
 - Headache
 - Blepharospasm
- The diagnosis can be confirmed by examining the cornea under **cobalt-blue light** following the application of **fluorescein**.
- If clinical examination is limited by pain, topical anaesthetic can be instilled.
- **Proxymetacaine** has been shown to produce the lowest pain score of all topical anaesthetics and regarded as the agent of choice for the examination of the injured eye.
- Corneal abrasions appear green under florescent light after the application of fluorescein.

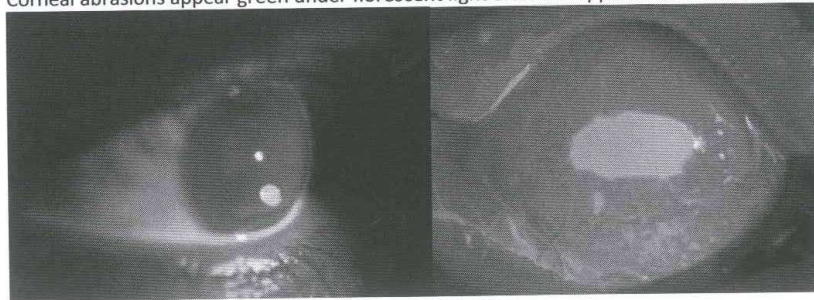


Figure 1.29.7. Corneal abrasion

- **Management:**
 - **Topical analgesics:** Topical non-steroidal anti-inflammatory drugs (NSAIDs) such as diclofenac and ketorolac have been shown to be modestly useful analgesics in corneal abrasion.
 - **Topical antibiotics**
 - There is no good published evidence that eye ointment is better than eye drops for preventing infection following corneal abrasion. However, expert consensus is that **eye ointments** are preferred because they are thought to be more lubricating.
 - In patients who wear contact lenses an **anti-pseudomonal antibiotic** (e.g. **gentamicin/ofloxacin/ciprofloxacin**) should be used and contact lens use discontinued. It is recommended that contact lenses are avoided until the antibiotic course is completed and the abrasion is healed.

Notes:

- **Eye pads:** Eye pads do not speed up recovery from a corneal abrasion and may worsen the pain and affect vision.
- **Mydriatics:** Mydriatics are no longer recommended for the treatment of pain in patients with corneal abrasions.
- **Topical corticosteroids:** Topical corticosteroids should not be used in the management of corneal abrasions.
- **Topical anaesthetics:** Topical anaesthetics slow healing and aggravate associated keratitis in corneal abrasion
- **Lubricants:** there is no evidence to support the use of lubricants in the treatment of corneal abrasions
- **Tetanus prophylaxis:** Routine tetanus prophylaxis is not recommended for corneal abrasion

2. CORNEAL FOREIGN BODY (INCLUDING RUST RING)

- Small metallic foreign bodies can come into contact with the eyes, most commonly when someone is drilling or grinding a metal surface.
- Special attention should be paid to the identification of a corneal rust ring. Iron in its neutral form is relatively insoluble in the corneal layers.
- However, over time a metallic foreign body's surface oxidises and diffuses into the stroma. A rust ring is then formed by the combination of oxidised iron and cellular infiltrate at the level of the superficial stroma.
- A rust ring can lead to **permanent corneal staining, chronic inflammation, corneal vascularisation and necrosis** and therefore should be removed within a few days of it being identified.



Figure 1.29.8. Corneal foreign body

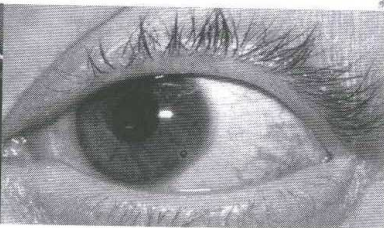
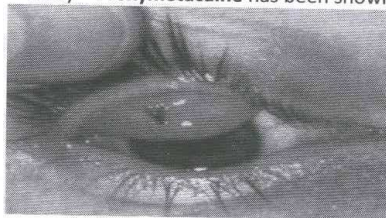


Figure 1.29.9. Corneal rust ring

• ANGLE GRINDING

- Patients will not always recall a foreign body having entered the eye so it is important to have a high index of suspicion and examine for a conjunctival or corneal foreign body if a patient presents with an uncomfortable red eye.
- Local anaesthetic may be needed both to examine the eye and to remove any foreign body – **Proxymetacaine** has been shown to be the optimal agent.



- If there is a history of a possible foreign body entering the eye and it cannot be seen then **the eyelid must be everted** to exclude a subtarsal foreign body, provided a penetrating injury is not suspected.
- If a subtarsal foreign body is present, it is easily removed using a cotton bud.
- Also, where the history is of a high velocity foreign body (e.g. metallic fragment from angle grinding or hammering a metal chisel) the possibility of a penetrating injury with intraocular foreign body must be considered.
- **ED MANAGEMENT**
 - **Instillation of local anaesthetic**
 - Small loose conjunctival foreign bodies can be **washed out with water or removed with a cotton bud.**
 - If the foreign body is adherent or embedded in the cornea, a **needle may be used to lift it out of the cornea.** This must be done either using a slit lamp or loupes to ensure accuracy and minimal damage to the cornea.
 - Once the foreign body has been removed any remaining epithelial defect can be treated as an abrasion.
 - Rust rings can be removed either **by a needle or by ophthalmic burr.**
 - It may be easier to remove rust rings 2 -3 days after presentation as local necrosis will separate the rust ring from the corneal epithelium.
- If there is any doubt, these patients should be **referred to an ophthalmologist.**

3. ULTRAVIOLET KERATITIS

- This condition arises from intense or prolonged exposure of the cornea to ultraviolet light, most commonly from welding (arc eye), sunlamps or reflected sunlight from snow (snow-blindness).
- The ultraviolet light irritates the corneal epithelium, triggering an inflammatory response with oedema and congestion developing into a superficial keratitis.
- Ultraviolet light exposure can cause **superficial keratitis.**
- Welders, skiers and people who use sunbeds without eye protection are most commonly affected.
- Symptoms occur several hours after exposure and include blepharospasm, photophobia, watering and pain.
- Local anaesthetic, whilst providing good relief from pain, allows for examination with fluorescein staining.
- The most common finding on examination is **generalised conjunctival injection** with multiple punctate lesions on the cornea.
- **ED MANAGEMENT**
 - The mainstay of management of an ultraviolet burn to the cornea is **pain relief**; Topical and oral analgesics such as **NSAIDs**
 - A **mydriatic** may be helpful for photophobia: **single dose of cyclopentolate** instilled in to ED will dilate the pupil for up to 24 hours, by which time the ultraviolet burn will normally have healed. If a mydriatic is used, the patient must be warned about blurring of vision and advised not to drive until the eye(s) have returned to normal.
 - A **topical antibiotic** is appropriate to prevent infection and lubricate the eye.
 - *Do not be tempted to discharge a patient with local anaesthetic drop as corneal healing will be delayed.*

4. CHEMICAL EYE INJURIES

- Chemical injuries represent 7-10% of all eye injuries presenting to the Emergency Department.
- Acid and alkaline solutions may cause corneal burns as can aerosol preparations such as **pepper spray** or **CS spray**.
- Chemical burns by alkaline solutions have the worst prognosis because they are able to penetrate the tissues quickly, whereas acidic solutions cause more superficial injuries.
- One should be alert in cases of a painless eye as there may be severe contamination.
- In the acute stage, chemical burns induce epithelial defects, corneal oedema, and ischaemic necrosis of the limbus, conjunctiva, iris and ciliary body.
- Patients usually present with a history of being splashed or sprayed in the eye(s), either accidentally or with a liquid chemical or CS spray or pepper spray.
- Assessment is usually very difficult due to reflex tearing and blepharospasm.
- The use of a local anaesthetic will ease pain and aid assessment which may need to follow the immediate priority of **eye irrigation for liquid chemicals**.

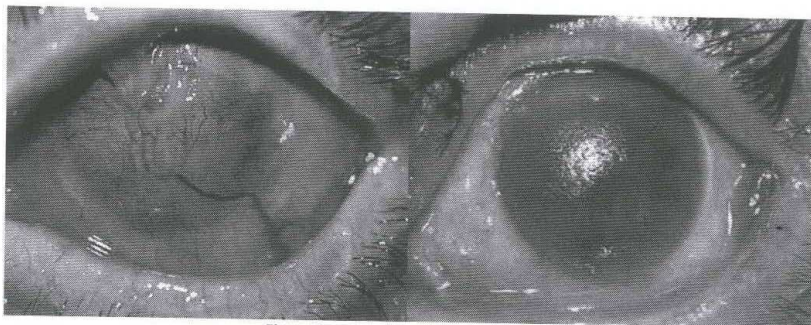


Figure 1.29.10. Chemical Eye injury

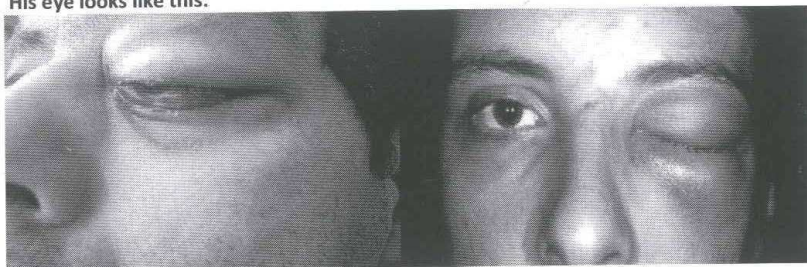
• ED MANAGEMENT:

- The recommended treatment for liquid chemical splashes to the eye is prompt **irrigation with copious amounts of water or normal saline**.
- **Litmus paper** should be used to judge the response to irrigation, which can be stopped when the pH returns to neutral (pH 7.0-7.3).
- Following irrigation, the eye(s) should be stained with **fluorescein**.
- If fluorescein uptake is demonstrated, referral to an ophthalmologist is recommended although local policy should be followed.
- **CS Spray or o-chlorobenzylidene malonitrile** is largely in use by police forces as a chemical incapacitant spray.
- Irrigation can worsen the symptoms as CS is highly soluble in water.
- Expert opinion suggests that the best way to manage corneal injuries resulting from CS spray is to **place the patient in a well-ventilated area and use a fan to blow air directly across the eyes**, ensuring no cross contamination occurs with others in the vicinity.

IV. EYE INFECTIONS

1. POST-SEPTAL (ORBITAL) CELLULITIS

- A 38 year-old professional fisherman presents to your emergency department after returning to shore from a 3 week trip. Over the past week he has had progressively worsening left eye symptoms and is now feeling quite unwell.
- He has been having fevers, has a constant headache and his left eye has become swollen.
- He reports a history of double vision but now he has trouble seeing anything out of his left eye. Any movement of the eye causes significant pain.
- His eye looks like this:



Q1. What is the likely diagnosis?

- **Orbital/ Post-septal cellulitis**
 - Features present in the image include:
 - Eyelid edema and erythema
 - Chemosis and an engorged conjunctiva
 - Proptosis

Q2. What features should be assessed when taking the history?

- **History**
 - **Symptoms:** Red Eye, Pain, Blurred Vision, Double Vision, Eyelid Swelling, Nasal Congestion, Sinus Headache/ Pressure, Tooth Pain, Periorbital Pain or Hypesthesia.
 - **Consider possible underlying causes:** Trauma, Surgery, ENT or Systemic Infection, Diabetes Mellitus, and Immunosuppression.

Q3. What features should be looked for on examination?

- **Examination**
 - **Visual acuity:** May be reduced in severe cases due to optic nerve stretch or compression
 - **External exam:**
 - **Eyelids:** Eyelid Edema, Erythema, Warmth, Tenderness
 - **Conjunctiva:** chemosis and injection
 - **Proptosis**
 - **Other:** Purulent discharge and decreased periorbital sensation may be present
 - **Extraocular eye movements:** Restricted ocular motility with pain on attempted eye movement.
 - **Pupils:** RAPD may be present in severe cases due to optic nerve stretch or compression
 - **Fundoscopy:** Retinal venous congestion and optic disc edema in severe cases.
 - **General exam:** Fever; and in severe and progressive disease altered mental state and meningism may occur.

Q4. What causative organisms are usually responsible for this condition in the different settings in which it can occur?

- **Adults:** Staphylococcus species, Streptococcus species, Bacteroides
- **Children:** Staphylococcus species, Streptococcus species, Haemophilus influenzae (rarely in vaccinated children)
- **Post-traumatic:** Gram-negative bacteria
- **Dental abscess:** mixed, aggressive aerobes and anaerobes
- **Immunocompromised or diabetes mellitus:** consider fungi, e.g. mucormycosis, zygomycosis, aspergillosis.

Q5. What investigations are required when considering this diagnosis?

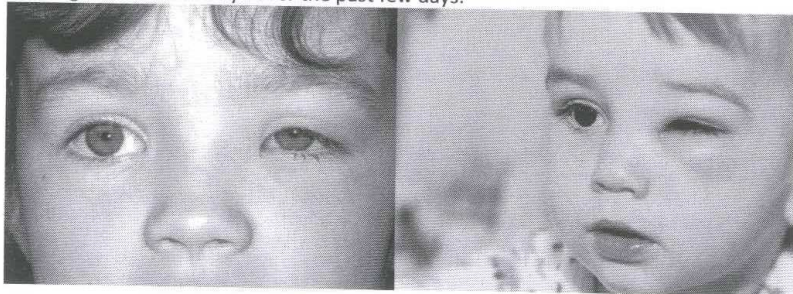
- **Laboratory:** FBC, blood cultures, wound swabs, consider the need for lumbar puncture.
- **CT scan of the orbits and sinuses:** Confirms the diagnosis and helps to exclude cavernous sinus thrombosis, orbital or subperiosteal abscesses, paranasal sinus disease and foreign bodies.

Q6. What is the appropriate management?

- Referral to ophthalmology for admission to hospital
- Consider consultant the following:
 - **Neurology:** if suspected CNS infection
 - **ENT:** if drainage of the sinuses is needed
 - **Oral/Maxillofacial surgeons:** if emergency dental extraction is needed
- **Therapeutic Guidelines:** Ceftriaxone 1-2 g IV bd + Metronidazole 500 mg IV, 8-hourly.
 - Anaerobic cover may be required (e.g. metronidazole), for instance, if a dental cause is suspected.
 - If MRSA is suspected consult an infectious disease specialist and consider treatment with vancomycin.
- **Other treatments may be required:**
 - **Analgesia**
 - **Nasal decongestant sprays** as needed for up to 3 days.
 - **Erythromycin ointment** qid: for corneal exposure and chemosis if there is severe proptosis.
 - **Canthotomy/cantholysis:** may be required if the orbit is tight, optic neuropathy is present or the IOP is severely elevated.
 - **Abscess drainage**

2. PRE-SEPTAL (PERIORBITAL) CELLULITIS

- Your next patient is a child who is systemically well. He has developed redness and swelling around his left eye over the past few days:



Q7. What is the likely diagnosis?

- Periorbital/ pre-septal cellulitis

Q8. What are the clinical features of this condition, and how is it distinguished from the fisherman's diagnosis?

- Periorbital (or preseptal) cellulitis is a soft-tissue infection of the eyelids that does not extend **past the orbital septum posteriorly**. It causes eyelid and periorbital edema, redness, and discomfort.
- The ocular exam should be essentially normal:
 - Normal visual acuity
 - FROEM without significant discomfort
 - Absence of proptosis
- Sometimes the clinical distinction is unclear and imaging is necessary (e.g. CT orbits and sinuses).

Q9. What organisms cause this condition in children <5 years of age?

- Much the same as for orbital cellulitis:
 - Staphylococcus aureus
 - Streptococcus pneumoniae
 - Streptococcus anginosus/milleri group
 - Haemophilus influenzae type b (Hib) in the unvaccinated

Q10. What is the antibiotic treatment of this condition?

- Systemically well children <5 years of age:
 - Amoxycillin+Clavulanate for 7 days OR
 - Cephalexin 12.5 mg/kg orally, 6-hourly for 7 days
- Older children or adults or children with an infected wound or stye, etc:
 - Flucloxacillin 500 mg orally, 6-hourly for 7 days
 - (cephalexin and clindamycin are options in the setting of penicillin hypersensitivity)
- If systemically unwell it is best to treat and investigate for orbital cellulitis.

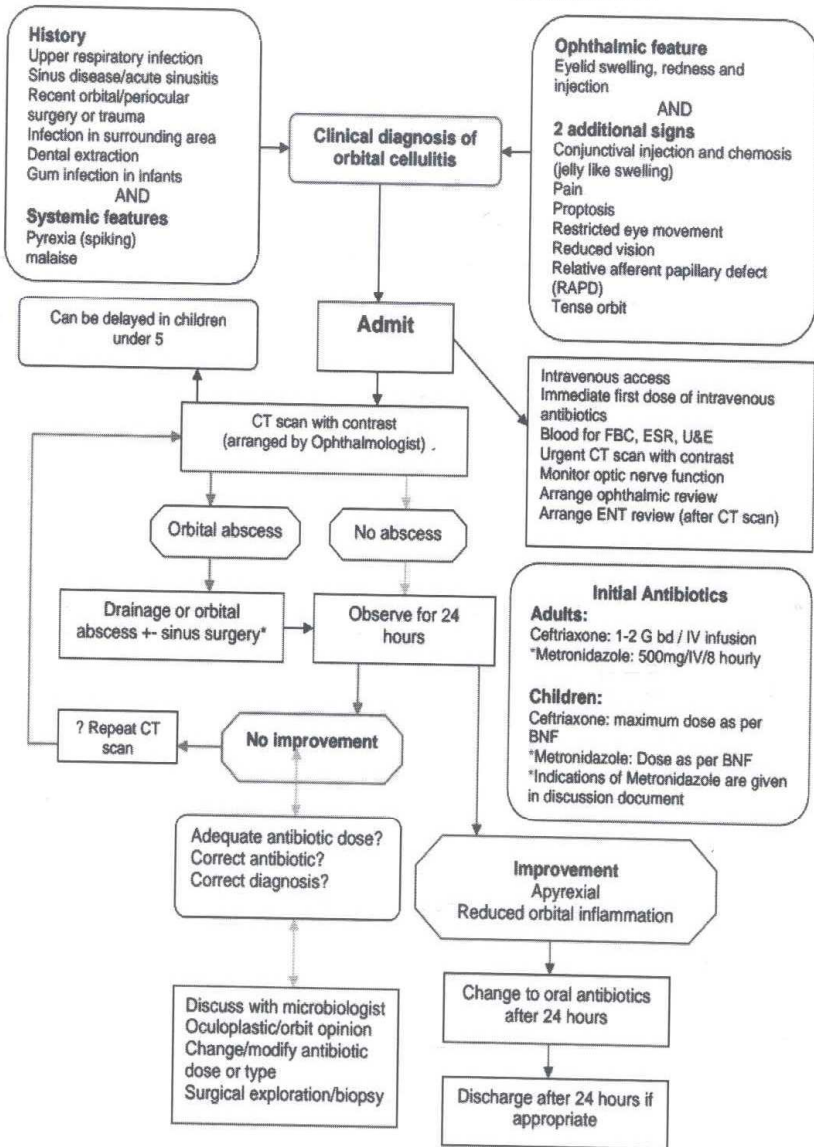
3. OPHTHALMIA NEONATORUM

- The commonest causative organism is *Chlamydia trachomatis*.
- Neonates of mothers with an untreated antepartum chlamydial infection have a 30-40% chance of developing chlamydial neonatal conjunctivitis postpartum.



- *Chlamydia trachomatis* typically produces conjunctivitis between **3 and 14 days** after birth. In contrast **Gonococcal Neonatal Conjunctivitis** typically occurs within the **first 5 days of life**, making it a less likely cause in a 7 day-old.
- Untreated cases may develop corneal ulceration, which may perforate resulting in corneal opacification and Staphyloma formation.

Management of Orbital Cellulitis in Adults and Children



CHAPTER 30. DENTAL EMERGENCIES**INTRODUCTION**

- Acute dental abscess usually occurs secondary to dental caries or following a dental procedure or trauma. Infection then may spread superficially into the tissues producing gingivitis or a dental abscess.
- Very occasionally infection spreads to the deep facial planes forming a **retropharyngeal abscess** or **Ludwig's angina**.
- Dental abscess is usually polymicrobial with numerous pathogen combinations being recognised, common pathogens include **streptococcus sp.** and **staphylococcus sp.**
- Staph. Aureus has been isolated in up to 15% of abscess cultures with the most common anaerobic species being **prevotella sp.** (10–87% of dentoalveolar abscesses).

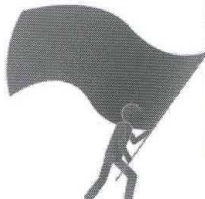
CLINICAL ASSESSMENT

- **Taking a History**

- o In the busy environment of the ED, history taking needs to be concise and focused, giving opportunity to highlight worrying features.
- o The salient points are:
 - When did the symptoms start?
 - Have antibiotics already been taken? A worsening condition in a patient already taking antibiotics is concerning
 - Has the patient seen a dentist? Most causes of mouth infection need definitive treatment by dealing with the cause – in general, ED care is a temporising measure only.
 - Is the patient systemically unwell? This indicates potential spread of the infection beyond the tooth and gum.
 - Is the patient immunocompromised? E.g. Diabetes, HIV, steroid use or general poor health.
 - Have you considered non-dental causes of 'tooth pain'?

- o **Examination**

- o When clinically assessing a patient a number of examinations need to be performed:
 - **Initial examination**
 - ABC assessment as, rarely, dental infection may be complicated by airway obstruction.



- The presence of any of the following may indicate actual or impending airway compromise and should prompt an urgent senior anaesthetic assessment:

- **Stridor**
- **Difficulty in breathing**
- **Dysphagia**
- **Dysphonia (alteration in the character of the voice)**

- **Examination of the face**

- **Visible facial swelling?** Describe it.
- **Does it extend into the neck?** This may indicate spreading infection.
- **Trismus** - an inability to open the mouth normally

- **Palpable lymph nodes**

- **Examination of the mouth**

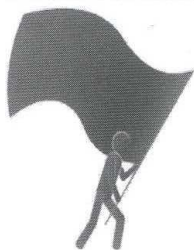
- Look at the teeth, what condition are they in? Is dental caries present?
- Are the gums red, bleeding, inflamed or swollen?
- Look in the sublingual space for swelling and redness these are signs of sublingual infection and potential airway compromise.
- Look at the pharynx is there evidence of swelling? this may indicate retropharyngeal spread of infection
- Feel in the mouth with a gloved finger for tenderness, swelling and/or fluctuance.

ED MANAGEMENT OF TOOTHACHE

- The vast majority of patients with toothache presenting to the ED should be managed with simple analgesia and advice to attend a dental practitioner as soon as possible.
- **Ibuprofen** being the drug of choice due to its low incidence of gastrointestinal side effects.
- **Paracetamol** also has a role to play both as an analgesic and anti-pyretic, either in addition to a NSAID or when NSAIDs are contra-indicated.
- **Opiates** should be reserved for adjunctive analgesia in situations where paracetamol and NSAIDs have failed to achieve adequate pain relief.

ED MANAGEMENT OF DENTAL INFECTION AND ABSCESS

- National antibiotic prescribing guidance for dental problems states that antimicrobials should be only given to patients with:
 - Symptoms or signs of systemic illness
 - High risk patients where complications are likely e.g. immunocompromised patients, diabetics
- Antibiotics are generally not indicated for otherwise healthy individuals or when there are no signs of spreading infection.
- They do not form the mainstay of treatment of local infection and early management of the source must be a priority
- **First-line treatment: Amoxicillin or Metronidazole for a total of 5 days.**
- If, after 48 hours, there is no improvement in systemic symptoms, consider changing to **second-line therapy**; either **adding metronidazole 400 mg twice daily** or changing to **co-amoxiclav 625 mg**.
- **Criteria of Maxillofacial referral/admission:**



- The presence of any of the following should prompt urgent referral to a maxillofacial specialist for consideration of admission:
 - *Evidence of significant systemic disturbance*
 - *Failure to control infection with antibiotics*
 - *Rapid spread of infection*
 - *Dysphagia*
 - *Dysphonia*
 - *Immunocompromised patients*
 - *General anaesthetic needed to drain an abscess*

1. LUDWIG'S ANGINA

- Ludwig's angina is an uncommon but important diagnosis not to be missed in patients attending the ED.
- It is a potentially life threatening complication of untreated dental infection.
- It is a rapidly progressing **submaxillary, submandibular, and sublingual necrotizing cellulitis** and can lead to **airway obstruction** and **death**.
- It requires an early diagnosis and treatment which may include an urgent surgical airway if the airway is compromised.
- 99% of cases of Ludwigs angina are odontogenic, anterior teeth often being the starting site for sublingual infection and, 2nd and 3rd molars are a starting point for submaxillary space infection.

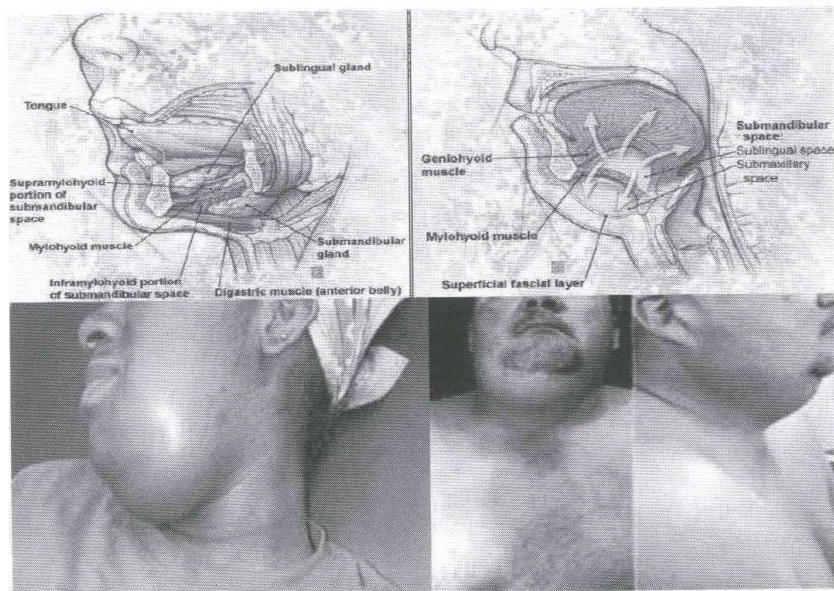


Figure 1.30.1. Ludwig's Angina

Presentation

- Dysphagia, neck pain and tooth pain,
- Signs such as tongue elevation and/or protrusion and neck swelling are all common.
- There may also be signs of airway obstruction such as stridor and dyspnoea.

Management of Ludwig's Angina

- **Swift recognition** and aggressive and early intervention is paramount.
- **Airway safety** is the primary concern
- Followed by administration of **intravenous antibiotics** and
- Consideration of **surgical drainage**.
- Early involvement of **Maxillofacial specialist, Anaesthetist** and **Otolaryngologist**

2. VINCENT'S ANGINA

- It is also known as **trench mouth** or **Acute Necrotising Ulcerative Gingivitis (ANUG)** and is defined as *an acute febrile, pseudo-membranous inflammation of the gingiva secondary to bacterial infection*.
- The aetiology is unclear but is thought to be due to superinfection with anaerobic bacteria such as **bacteroides**, **fusobacterium** and **spirochetes** which are normal mouth flora.
- It is now more commonly found in patients with immunodeficiency states such as malnutrition and HIV infection.

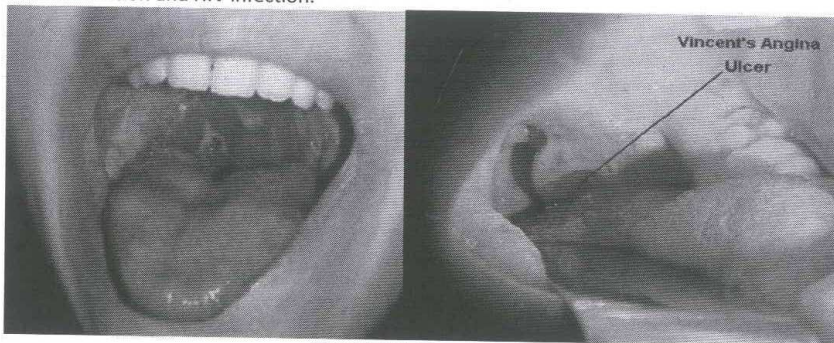


Figure 1.30.2. Vincent's angina

Presentation

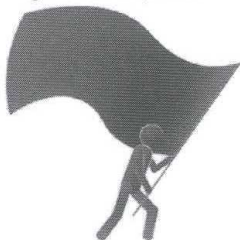
- The infection starts as inflammation, usually on one side of the mouth, which spreads along the gingival margins and may affect the pharynx and lips.
- This leads to gum atrophy, ulceration, enlarged lymph nodes and formation of a grey pseudo membrane, which may lead to it being confused with diphtheria.

MANAGEMENT OF VINCENT ANGINA IN ED

- **Chlorhexidine** mouth washes
- A 3-day course of **Metronidazole** or **Amoxicillin**
- Encouragement to **improve oral hygiene**
- Patients should be advised that they need urgent assessment and management by a dentist.

CHAPTER 31. E.N.T. EMERGENCIES**I. ACUTE SORE THROAT****INTRODUCTION**

- **Common Causes:**
 - Tonsillitis
 - Pharyngitis
 - Peritonsillar Abscess (quinsy)
 - Infectious mononucleosis
- **Less common and/or dangerous causes of a sore throat include:**
 - Lemierre's syndrome due to *Fusobacterium necrophorum*
 - Retropharyngeal abscess
 - Epiglottitis
 - Scarlet fever/ Diphtheria
 - Bacterial Tracheitis
 - Ludwig's Angina
 - Angioedema or anaphylaxis
 - Painful cervical lymphadenopathy
 - Trauma, e.g. foreign body or caustic ingestion
- Most sore throats have an unknown or viral aetiology but **Group A β -haemolytic streptococcus (GABHS)** is identified as the infecting agent in approximately 5-15% of all cases. GABHS infection may be complicated by significant sequelae such as **Rheumatic fever, peritonsillar abscess** (quinsy) and **post-streptococcal glomerulonephritis**.
- Although the minority of sore throats are caused by bacterial infection, almost 2/3 of patients are managed with a course of antibiotics.
- **Other less common bacterial causes of pharyngitis/ tonsillitis include:**
 - Group C and G strep
 - *Fusobacterium necrophorum*
 - *Neisseria gonorrhoea*
 - *Corynebacterium diphtheriae*
 - *Mycoplasma pneumoniae* and several chlamydial species
- Complications of GABHS infection are categorised into suppurative and non-suppurative:
 - **Suppurative complications:** otitis media, sinusitis and peritonsillar abscess.
 - **Non-suppurative complications:** rheumatic fever, post-streptococcal glomerulonephritis



- There are a number of **RED FLAG SYMPTOMS** and signs that should prompt the clinician to consider a more serious cause for a sore throat, including;
 - **Significant systemic upset**
 - **Severe pain**
 - **Stridor (airway obstruction)**
 - **Severe neck stiffness**
 - **Inability to swallow / drooling of saliva**
 - **Patient holding a tripod position**

- A patient with signs of potential or partial airway obstruction such as stridor, inability to swallow and holding a tripod position **must be assessed urgently by a senior anaesthetist and otolaryngologist.**

RISK STRATIFICATION

- To differentiate between viral causes and the potentially more serious GABHS infection, a number of tools have been developed to assess the probability of GABHS infection and therefore the need for antibiotic treatment.
- The most commonly used score is that developed by Centor and recently modified by McIsaac.

CENTOR/ MODIFIED MC ISAAC CLASSIFICATION

MCISAAC SCORE		POINTS	SUGGESTED MANAGEMENT
History of fever or $T^0 > 38^{\circ}\text{C}$	+1	-1 or 0	No culture or antibiotic
Absence of cough	+1	1	No culture or antibiotic
Tender anterior cervical lymphadenopathy	+1	2	Culture all treat those with +ve culture
Tonsillar swelling or exudates	+1	3	Culture all treat those with +ve culture
Age 3-15 yrs	+1	4 or 5	Treat with antibiotic
Age 15 to < 45yrs	0		
Age ≥ 45 years	-1		

INVESTIGATIONS

- **Rapid streptococcal antigen testing** is very accurate at identifying GABHS infection but sensitivity is poor and experience in EDs in the UK is very limited.
- A rising **antistreptolysin O titre** (ASOT) provides the gold standard criteria for immunologically significant GABHS infection. However it is impractical and unnecessary in the vast majority of cases.
- **Throat swabs**, although widely used, are reliant on correct technique and interpretation is complicated by asymptomatic carriers of GABHS.
- Other investigations which may be useful in patients with a sore throat include:
 - **Monospot and PaulBunnell tests** for infectious mononucleosis.
 - **Chest x ray** if respiratory infection is suspected
 - **Lateral soft tissue neck x ray** for retropharyngeal abscess and epiglottitis: An epiglottic width (widest anteroposterior diameter of the epiglottis) of >7 mm was found to have a sensitivity and specificity of 100% for the diagnosis of epiglottitis in one study.

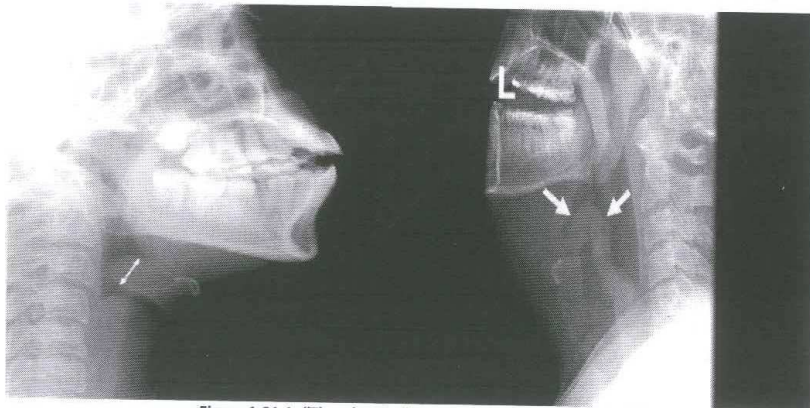


Figure 1.31.1. "Thumb print" sign on lateral xray of epiglottitis

MANAGEMENT OF SORE THROAT IN ED**1. TONSILLOPHARYNGITIS**

- **Current recommendation: No initial antibiotics are given** and the patient is **advised to return to their GP if their symptoms are not settling after a few days.**
- **Indications for Antibiotherapy:**
 - Marked systemic upset
 - An increased risk of complications:
 - **Immunosuppressed patients** e.g. diabetics or taking disease modifying anti-rheumatic drugs
 - **History of valvular heart disease**
 - **History of rheumatic fever**
 - An outbreak of GABHS infection within an institution (e.g. barracks / boarding school)
- If prescribing an antibiotic, **Phenoxymethylpenicillin is the standard agent recommended for a 10-day course.**
- In penicillin allergic patients, **Erythromycin or Clarithromycin** should be used.
- Other: **NSAIDs, Paracetamol, Pneumococcal vaccine and improved doctor-patient communication.** Simple non-antibiotic treatments such as NSAIDs and paracetamol are effective in patients with a sore throat and may have a greater positive effect than antibiotics alone.

2. SCARLET FEVER

- Scarlet fever is a GABHS exotoxin-mediated illness which occurs far more commonly in children.
- Other than standard antibiotic treatment for GABHS, consideration must also be made of **hydration status and intravenous fluid rehydration** may be required.



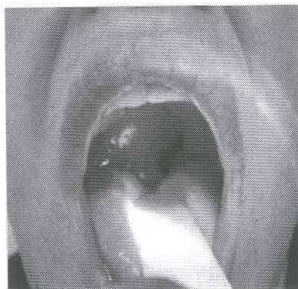
Figure 1.31.2. Strawberry tongue

3. PERITONSILLAR ABSCESS**SYMPTOMS**

- Progressively worsening sore throat, often localized to one side
- Fever
- Dysphagia
- Otagia
- Odynophagia

PHYSICAL EXAMINATION

- Erythematous, swollen tonsil
- Contralateral uvular deviation
- Trismus
- Edema of palatine tonsils
- Purulent exudate on tonsils
- Drooling
- Muffled, "hot potato" voice
- Cervical lymphadenopathy



- Uncomplicated peritonsillar abscess may be managed in the ED although it is common practice for patients to be referred to an ear, nose and throat (ENT) specialist due to a lack of familiarity with treatment techniques.
- Both **needle aspiration and incision and drainage techniques** may be used employed and have been found to be equally effective.
- The clinician must be aware of the potential complications of both the problem e.g. **Lemierres syndrome** (extension of infection involving the jugular vein) and its management e.g. **accidental puncture of the carotid artery**.

4. EPIGLOTTITIS

- Since the advent of Hib vaccination, this is now more commonly an infection affecting adults.
- The main complication of airway obstruction may be predicted by the presence of specific clinical features:
 - *Stridor*
 - *Muffled voice*
 - *Rapid clinical course*
 - *History of diabetes*
- **Routine intubation was unnecessary** as over 90% of patients recovered with a conservative watchful approach.
- Antibiotics – **IV Ceftriaxone 2g BD X 7days** and **Metronidazole** are recommended to cover the spectrum of organisms responsible.

5. RETROPHARYNGEAL ABSCESS

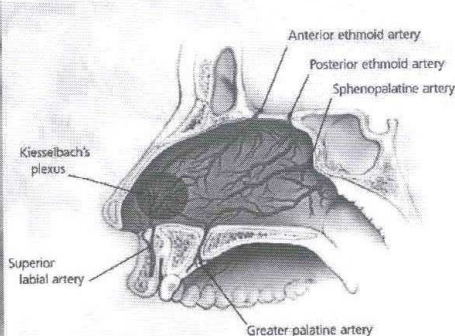
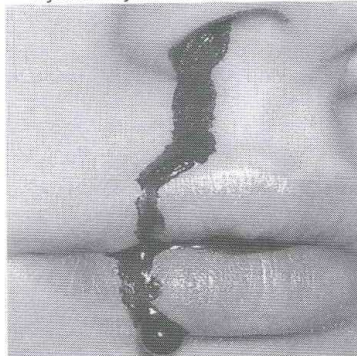
- Although very uncommon, a combination of *sore throat, fever, neck stiffness and stridor should alert the clinician to consider this diagnosis*.
- Swelling or oedema of the posterior pharynx should prompt a consideration of advanced airway care and an urgent ENT opinion.
- Mortality rates are high when complications such as airway obstruction and mediastinitis arise.
- Signs suggestive of potential airway obstruction are: **stridor, altered voice, inability to swallow saliva, tripod position >>> call ENT and Anaesthetist immediately.**
- **Sore Throat+ Fever+ Neck Stiffness+ Stridor= Retropharyngeal Abscess**

SIALOLITHIASIS

- It is a condition where a calcified stone (sialolith) forms within a salivary gland.
- Approximately 90% occur in the submandibular gland (**Wharton's duct**), with the majority of the remainder occurring within the parotid gland. Occasionally they can also occur in the sublingual gland or minor salivary glands.
- The sialolith obstructs the passage of saliva, which causes pain and swelling of the affected gland on eating. Pain is typically at its worst when salivary flow is high, immediately before and during eating, and then slowly subsides in the hour after.
- Bimanual palpation of the floor of the mouth may reveal a stone and occasionally the stone may actually be visible intra-orally at the duct orifice. If superimposed infection is present it may be possible to express pus from the gland.
- Acidic foods, such as lemon juice, can be used to stimulate salivary flow and may promote spontaneous expulsion of the stone.
- X-rays of the floor of the mouth can demonstrate the stone.
- Patients should be referred to ENT for removal of the stone.

II. EPISTAXIS

- A 34yo female presents to ED at 2am, post waking up with blood all over her pillow, and a continuous ooze of blood from her right nostril.
- On examination the patient is alert and oriented, BP 110/60, pulse 95, respiratory rate 22, SpO2 98% room air, and has no past medical history. The patient reports having a sinus infection of late which she's been using an antihistamine nasal spray to treat.



- Epistaxis is a frequent complaint
- 60% of the population will suffer from a nose bleed during their lifetime, and 6% will require medical attention.
- Majority of epistaxis occurs between the ages of **2-10** and **50-80 years old**.
- Epistaxis results from an interaction of factors that damage the nasal mucosal lining, affect the vessel walls, or alter the coagulability of the blood.
- Emergency physicians have a 90% success rate at treating epistaxis in emergency department, and only have to refer 10% to ENT for further assessment and management

CAUSES OF EPISTAXIS

Local trauma:	Coagulopathies	Vascular Abnormalities
<ul style="list-style-type: none"> ○ Nose picking ○ Facial trauma ○ Foreign bodies ○ Nasal or sinus infections ○ Nasal septum deviation 	<ul style="list-style-type: none"> ○ Von Willebrand disease, ○ Haemophilia A & B ○ Splenomegaly ○ Thrombocytopenia ○ Platelet disorders ○ Liver disease ○ Renal failure ○ Chronic alcohol abuse ○ AIDS 	<ul style="list-style-type: none"> ○ Sclerotic vessels ○ Hereditary haemorrhagic telangiectasia ○ Ateriovenous malformation ○ Neoplasm ○ Aneurysms ○ Septal perforation ○ Septal deviation ○ Endometriosis
Environmental	Iatrogenic	Medicinal
<ul style="list-style-type: none"> ○ Dry cold conditions (presentations increase during winter) ○ Prolonged inhalation of dry air (Oxygen) 	<ul style="list-style-type: none"> ○ Nasogastric tube insertion ○ Nasotracheal intubation 	<ul style="list-style-type: none"> ○ Anticoagulants: Aspirin, warfarin, platelet inhibitors ○ Topical corticosteroids and antihistamines ○ Solvent inhalation (huffing) ○ Snorting cocaine

• Hypertension:

- Controversial topic and is often misunderstood in epistaxis
- Hypertension is rarely a direct cause of epistaxis
- Epistaxis is however more common in hypertensive patients this is postulated to be caused from long standing hypertension causing vascular fragility of the blood vessels.
- Epistaxis in patients presenting to ED, will generally have an associated anxiety that will increase blood pressure. Despite multiple causes for epistaxis, literature shows that in 85% of cases no causes in found.

ANATOMY AND PHYSIOLOGY OF EPISTAXIS:

- The nose is supplied with an extensive vasculature with multiple anastomosis.
- 90% of epistaxis occurs in the anterior nasal septum, from **Little's area** which contains the **Kiesselbach plexus of vessels (LEGS Vessels)**.
- The other 10% occur posteriorly, along the nasal septum or lateral nasal wall.
- The blood supply of the nasal septum is from the **internal carotid** through the **anterior and Ethmoidal arteries**, and from **external carotid** through the **Greater palatine, Sphenopalatine and superior Labial arteries**.

ASSESSMENT OF THE PATIENT PRESENTING WITH EPISTAXIS:

• History:

- Obtain the following:
 - Laterality, duration, frequency, Severity, estimated blood loss
 - Any contributing or inciting factors
 - Family history or bleeding disorder, Past medical history
 - Current medications

• Physical Examination:

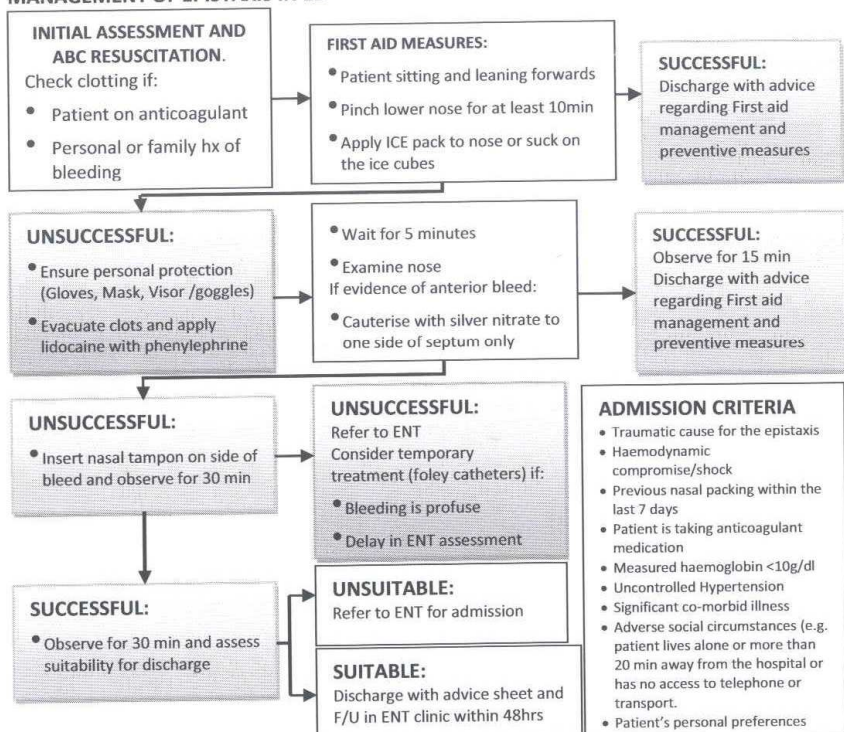
- Focus on trying to identify if the bleed is coming anteriorly or posteriorly.
- Suctioning or blowing of the nose to clear away clots, and application of topical vasoconstrictors or anaesthetics will help with visualisation.
- Gently insert nasal speculum and spread naris vertically, a good light source will be also required to assist visualisation of bleeding area.
- A posterior source of bleeding is suggested by failure to visualise an anterior source, bleeding from both nares, and the visualisation of blood in the posterior pharynx.

INVESTIGATIONS

- **FBC, U&E, LFT** (renal failure = U&E, chronic alcohol abuse = LFTs)
- **INR:** Patients taking warfarin
- **Coagulation:** only of benefit in patients with a known coagulopathy or chronic liver disease, and should not be routine in patients presenting with epistaxis.
- Radiological investigations have little role in the management of epistaxis,
- **CT scan** is indicated if neoplasm suspected, and would generally be arranged post consultation with your ENT specialist.

FIRST AID MEASURES TO STEM NASAL BLEEDING:

- **Lean the patient forwards in an upright position;** Encourage the patient to **spit out** any blood passing into the throat
- **Firmly pinch the soft part of the nose** compressing the nostrils for at least 10 minutes. If unable to comply then an alternative technique is to **ask a relative or staff member** or apply **swimmers nose clip**.
- **Use of ice:** to the neck or forehead; sucking on an ice cube or applying an ice pack ice directly to the nose may help

MANAGEMENT OF EPISTAXIS IN ED

- **Equipment and Personal Protection**

- Gloves, mask and visor
- Essential items for managing epistaxis: light source, Suction apparatus
- A combination anaesthetic and vasoconstrictor agent: lidocaine with phenylephrine.
- Nasal speculum

- **Nasal Cautey:** silver nitrate application stick or equipment for electrocautery

- **Do not cauterize both side of the nasal septum:** There is a risk of septal perforation due to decreased vascular supply from the perichondrium

- **Topical Treatment**

- In children, it is normally the case that adequate first aid measures will stop bleeding.
- Children with recurrent nose bleeds and nasal crusting should be treated with **topical nasal antiseptic (Naseptin) cream applied twice daily for 4 weeks.**
- In the presence of a visible vessel on the septum, cauterisation with silver nitrate is recommended.
- *Topical antiseptic cream is as effective as silver nitrate cautery in preventing further nosebleeds in children with recurrent epistaxis.*

- **Nasal Packing:** ribbon gauze packs

Insertion of foley catheters to stop uncontrolled posterior bleeding is a technique of last resort when immediate specialist help is unavailable.

ADMISSION CRITERIA AFTER ANTERIOR NASAL PACKING

- Traumatic cause for the epistaxis
- Haemodynamic compromise or shock
- Previous nasal packing within the last 7 days
- Patient is taking anticoagulant medication
- Measured haemoglobin less than 10 g/dl
- Uncontrolled hypertension
- Significant co-morbid illness
- Adverse social circumstances (e.g. the patient lives alone or more than 20 minutes away from the hospital or has no access to telephone or transport)
- Patients personal preference

PROGNOSIS & FOLLOWUP STRATEGIES

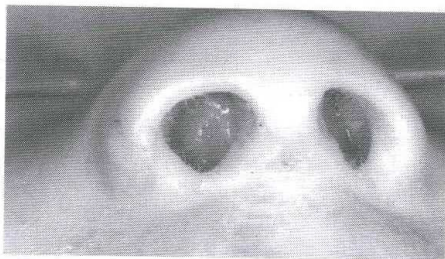
- No follow-up is necessary for patients in whom the epistaxis has either stopped spontaneously or by 1st aid measures or cautery alone.
- However, it is important to provide advice to prevent recurrence of the nosebleed and first aid measures for future episodes.

ADVICE TO PREVENT RECURRENCE OF EPISTAXIS

- **Avoidance of:**
 - Blowing the nose for one week.
 - Sneezing through the nose: keep the mouth open.
 - Hot and spicy drinks and food, including alcohol for two days.
 - Heavy lifting, straining or bending over.
 - Vigorous activities for one week.
 - Picking the nose.
- For those patients who have an anterior nasal pack, it should be left in place for **24-48 hours** and follow-up arranged with the ENT department for its removal and further assessment.
- *Routine antibiotic cover is unnecessary for patients with an anterior pack in place for less than 48 hours.*

SEPTAL HAEMATOMA

- Blood has collected in the cavity between the **cartilage and the supporting perichondrium**.
- This is typically caused by a shearing force stripping the perichondrium away from the underlying cartilage.
- Septal haematomas should be **drained** by needle aspiration to avoid complications occurring.
- Following drainage the nose should be **firmly packed** to avoid re-accumulation of the haematoma and broad spectrum antibiotics should be given.
- Left untreated septal haematomas are associated with the following complications:
 - *Septal abscess formation*
 - *Cartilage necrosis*
 - *Collapse of nasal bridge ('saddle nose')*
- Following treatment the patient should be **followed up in the ENT clinic in one week**.

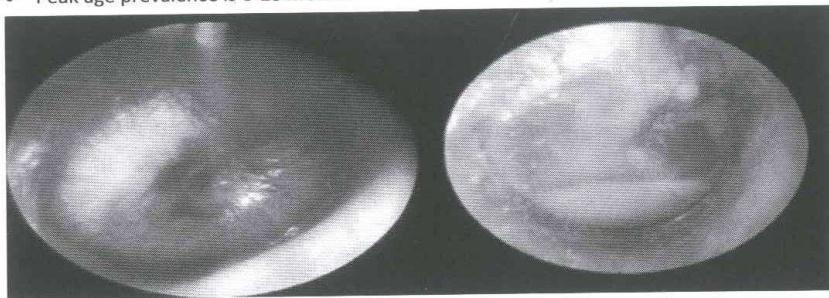


III. OTITIS

1. ACUTE OTITIS MEDIA IN CHILDREN

DEFINITION

- Acute otitis media is the presence of a middle ear effusion accompanied by rapid onset of one of otalgia, otorrhoea, irritability in an infant or toddler, or fever.
- Acute otitis media (AOM) is a common problem in early childhood with 2/3 of children experiencing at least one episode by age 3, and 90% have at least one episode by school entry.
- Peak age prevalence is **6-18 months**



- Acute otitis media (AOM): images demonstrate a bulging, erythematous tympanic membrane with loss of the light reflex.

AETIOLOGY

- *Streptococcus pneumoniae* (35%)
- Viral (25%)
- Non-typable strains of *Haemophilus influenzae* (25%)
- *Moraxella catarrhalis* (15%)
- The commonest causative organism of AOM is *Streptococcus pneumoniae*, which is responsible for 30-40% of cases.

INDICATIONS TO ADMINISTER ANTIBIOTICS FOR AOM

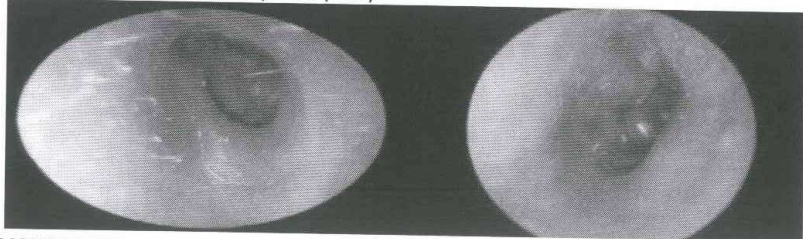
- *Children under 2 years with bilateral infection*
- *Presence of purulent discharge from ear*
- *If systemically unwell (e.g. fever and vomiting)*
- *Recurrent infections*
- **Amoxicillin** is the recommended first-line antibiotic for AOM, where antibiotics are indicated. **Five days** treatment at the following doses is sufficient for uncomplicated ear infections in children. The doses are as follows:
 - **Neonate (7-28 days):** 30mg/kg TDS
 - **1 month-1 yr:** 125mg TDS
 - **1-5 years:** 250mg TDS
 - **5-18 years:** 500mg TDS
- If the patient is penicillin allergic then **Erythromycin** (or suitable macrolide antibiotic alternative) should be prescribed for **5 days**. The doses are as follows:
 - **<2 years:** 125mg QDS
 - **2-8 years:** 250mg QDS
 - **8-18 years:** 250-500mg QDS

- **POTENTIAL COMPLICATIONS OF AOM**

- *Chronic secretory otitis media*
- *Conductive hearing loss*
- *Tympanic membrane perforation*
- *Acute mastoiditis*
- *Meningitis*
- *Facial nerve palsy*
- *Brain and Dural abscesses*
- *Endocarditis*

2. OTITIS EXTERNA

- It is infection and inflammation of the ear canal. Common symptoms include pain, itching and discharge from the ear. Otoscopy will reveal erythema of the ear canal with pus and debris present. Various conditions can predispose to otitis externa including skin conditions, such as psoriasis and eczema. It is also more prevalent in people that have regular exposure to water in the ear canal, such as swimmers (**Swimmer's ear**).
- **RISK FACTORS**
 - Swimming
 - Congenital narrowing of the ear canal
 - Foreign object in the ear canal e.g. cotton bud or hearing aid
 - Trauma to the ear canal e.g. overly vigorous cleaning
 - Skin conditions e.g. eczema or psoriasis
- **The commonest causative organisms are:**
 - *Pseudomonas aeruginosa* (50%)
 - *Staphylococcus aureus* (23%)
 - Gram negative bacteria e.g. *E.coli* (12%)
 - *Aspergillus* and *Candida* species (12%)



ED MANAGEMENT OF OTITIS EXTERNA

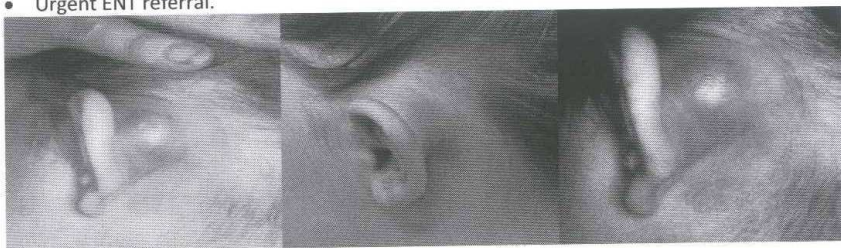
- Keep the ear dry and advise against inserting anything into the ear.
- Simple analgesia.
- Topical ear drops e.g. combined corticosteroid and antibiotic.
- An aminoglycoside is contraindicated if the tympanic membrane is perforated.
- Aural toilet \pm wick insertion if extensive debris.
- A referral to the on call ENT team would be warranted if any of the following are present:
 - *Concurrent skin infection e.g. erysipelas or cellulitis*
 - *Presence of necrotizing otitis externa (osteomyelitis)*
 - *Failure to respond to first line treatment*
 - *Aural toilet required*
 - *History of chronic ear condition*

IV. MASTOIDITIS

- Mastoiditis is an infection of the mastoid process of the temporal bone. It is fortunately an uncommon complication of acute otitis media but can lead to intracranial infection.
- Clinical features that help identify mastoiditis
 - Erythema, swelling, and tenderness over the mastoid process.
 - Displacement of the pinna forwards and outwards.
 - Narrowing of the external auditory canal.
 - Failure of treatment in acute otitis media.

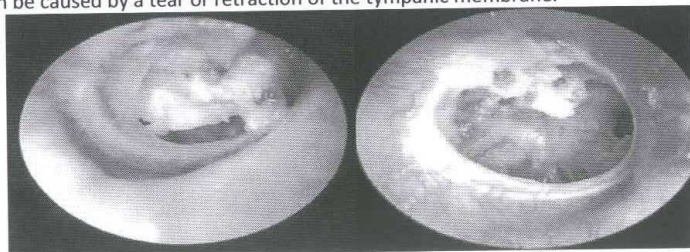
ED MANAGEMENT OF MASTOIDITIS

- Intravenous broad-spectrum antibiotics.
- Urgent ENT referral.



V. CHOLESTEATOMA

- Cholesteatoma is an erosive disorder of the middle ear and mastoid, which can lead to life-threatening intracranial infection.
- It can be caused by a tear or retraction of the tympanic membrane.



SIGNS AND SYMPTOMS

- Painless otorrhea, either unremitting or frequently recurrent.
- Conductive hearing loss
- Dizziness: Relatively uncommon
- Drainage and granulation tissue in the ear canal and middle ear: Unresponsive to antimicrobial therapy
- Occasionally, cholesteatoma initially presents with symptoms of CNS complications, including the following:
 - *Sigmoid sinus thrombosis*
 - *Epidural abscess*
 - *Meningitis*
- Patient should be referred urgently to ENT for a CT scan and surgical removal of the lesion.

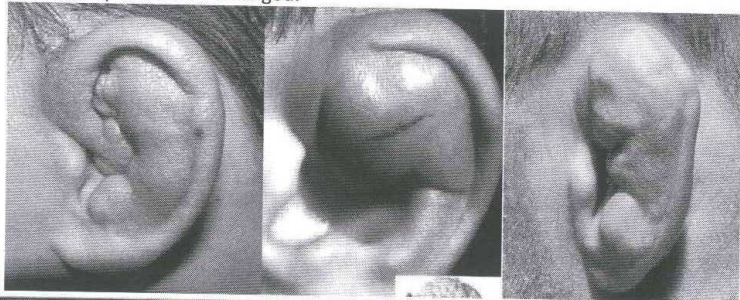
VI. EAR INJURIES

1. TYMPANIC PERFORATION

- Traumatic tympanic perforation may be caused by barotrauma, direct penetrating injury (e.g. cotton bud), or following a base of skull fracture.
- The patient experiences pain, reduced hearing, and sometimes a bloody discharge.
- Most perforations will heal spontaneously and the patient should be advised to keep the ear clean and dry. They should not put anything into the auditory canal.
- GP follow-up should be arranged to ensure adequate healing.

2. AURICULAR HAEMATOMA ('CAULIFLOWER EAR')

- Blunt trauma to the external ear can result in a haematoma forming under the perichondrium. This separates the cartilage, which is avascular, from the perichondrium, which supplies it, resulting in necrosis.
- The haematoma should be aspirated acutely and a firm dressing applied over the ear and around the head.
- ENT follow-up should be arranged.



PANCOAST TUMOUR

- It is a tumour that occurs at the apex of the lung, most are non-small cell cancers.
- **The growing tumour can cause compression of a number of nearby structures including:**
 - *Recurrent laryngeal nerve (causing hoarseness)*
 - *The sympathetic ganglion (causing Horner's syndrome)*
 - *Phrenic nerve*
 - *Brachiocephalic vein*
 - *Subclavian artery*
 - *Superior vena cava*
- Approximately 5-15% of lung cancer patients develop hoarseness as a consequence of recurrent laryngeal nerve compression and the left side is most commonly affected.
- Horner's syndrome is a combination of symptoms that arises when the sympathetic ganglion is damaged.
- The following clinical **features classically** occur on the same side as the lesion:
 - **Miosis**
 - **Anhidrosis**
 - **Ptosis**
 - **Enophthalmos**

CHAPTER 32. PSYCHIATRIC EMERGENCIES**I. MENTAL HEALTH ACT****INTRODUCTION**

- The Act sets out five principles that are designed to regulate decisions made under the legislation (any three will score a mark each):
 - A person must be assumed to have capacity unless it is established that he or she lacks capacity
 - A person is not to be treated as unable to make a decision unless all practicable steps have been taken to help him or her
 - A person is not to be treated as unable to make a decision just because he or she makes an unwise decision
 - All decisions must be made in the incapacitated person's best interests
 - Decisions made must be least restrictive of the individual's fundamental rights or freedoms
- The most important parts are **2, 3, 4, 5 & 135 and 136.**

1. SECTION 2

- **Section 2** aka an **Assessment Order**— allows a patient to be **sectioned for up to 28 days**. Must be **signed by 2 doctors and an ASW** (ASW – approved social worker).
- These professionals must agree that the patient is mentally unwell, and they require a **full assessment** in a **psychiatric** setting.
- The patient must have been examined by the two doctors within 5 days of each other.
- The two doctors cannot be employed by the same organisation.
- One of the doctors has to have previously known the patient.
- It allows patients to be treated against their will, as they are seen to be mentally unstable. **Cannot be renewed**
- Commonly the doctors involved are the **patient's GP, and a psychiatrist**.
- Type of mental disorder that the patient is thought to be suffering from does not have to be disclosed. Treatment can be given against the patient's will – as this is considered part of the assessment process.

2. SECTION 3

- **Section 3** aka **treatment orders** – same as section 2, but **for 6 months**.
- The ASW must seek the consent of the nearest relative, and the patient cannot be detained if this relative objects.
- **Can be renewed** for 6 months or even sometimes for a year
- The doctor has to state the category of mental illness the patient is thought to be suffering from (e.g. mental illness, psychosis, mental impairment)
- The majority of 'sectionings' are treatment orders
- **Treatment can be given** – but after 3 months, either:
 - The patient has to consent to treatment. A third doctor has to review the patient and give their consent for treatment to be given
- To be discharged from sections 2 & 3, the patient has to be discharged by one of:
 - The RMO (registered medical officer)
 - Hospital managers
- The nearest relative can ask for discharge; however in practice it is unlikely that patients will be discharged before the sectioning is over.

- **Appeal** – patient may appeal to mental health review tribunal
- **Section 2 appeal** – must be made within **14 days**
- **Section 3 appeal** – must be made within **6 months**

3. SECTION 4

- Requires support of one medical practitioner and allows **Emergency detention for 72 hours for assessment**. The application can be made by an approved mental health practitioner or the nearest relative. **Renewal of section 4 is not possible** but it may be converted within 3 days of admission to a **section 2** by means of a second medical recommendation.

4. SECTION 5

A. SECTION 5(2)

- A Section 5(2) is known as the **Doctor's holding power**. The doctor in charge of the patient's care must write a report explaining the detention and why informal treatment is inappropriate.
- A s5(2) can be used both in a mental health hospital and a general hospital.
- Under a s5(2), patient can be held for up to 72 hours. This is not renewable.
- Patient must be assessed as quickly as possible by an Approved Mental Health Professional (AMHP) and doctors for possible admission under the Mental Health Act.
- *Under sections 5(2) and 5(4), patient can **refuse treatment** and **must give consent** for any treatment that is given to him/her.*
- *Unless the patient:*
 - *Does not have the capacity to make a decision about treatment and the treatment is in the patient's best interests.*
 - *Needs treatment in an emergency to prevent serious harm to himself or others.*

B. SECTION 5 (4)

- A section 5(4) is known as the **Nurse's Holding Power**.
- This power can only be used:
 - *To prevent patient from leaving hospital for his/her own health or safety or for the protection of others*
 - *When it is not possible to get a Doctor who can section the patient under s5(2)*
- Under s5(4), patient can be held up to 6 hours. This is not renewable.
- The holding power ends as soon as a doctor arrives. The doctor may transfer the patient onto a s5(2) or you may continue as a voluntary patient.
- If the patient needs to be detained under a section 2 or 3, an assessment by an Approved Mental Health Professional (AMHP) and doctors must be arranged as quickly as possible.

5. SECTION 135

- A police constable may enter the patient's premises and remove a person to a **'place of safety'** for up to **72 hours**. Can use force if need be. Can only be used if a social worker has obtained a warrant. **Cannot treat against the patient's will.**

6. SECTION 136

- Section 136 of the MHA allows a police officer to remove someone who appears to be suffering from a mental health disorder to a place of safety.
- This allows detention for **72 hours** and allows the patient to be assessed by a medical practitioner. Convert to s2 or s3 if admission is required.

II. DELIBERATE SELF-HARM**A GUIDELINE FOR ED STAFF****• GENERAL PRINCIPLES**

- o Patients who harm themselves have high rates of mental disorder, life stress and have an increased risk of further self-harm and suicide.
- o **All** patients presenting to the ED following self-harm should have a brief mental health assessment by ED staff and should be referred to a trained mental health professional for assessment at the earliest possible opportunity.

• IMMEDIATE TRIAGE

- o Patients should be triaged on arrival with the mental health triage scale in addition to the standard triage. Staff should be aware of ongoing availability of means of repetition (e.g. tablets, weapon on person) and deal with this risk accordingly.

• ED DOCTOR ASSESSMENT

- o In addition to necessary medical assessment and management, the ED Doctor should also consider the following:
 - Is the patient physically fit to wait?
 - Is there obvious severe emotional distress?
 - Is the person actively suicidal?
 - Is the person likely to wait for medical treatment and further mental health assessment?
 - Does the patient have mental capacity?

• WHEN A PATIENT FOLLOWING SELF-HARM REFUSES TREATMENT

- o Remember that the **MHA cannot be used in the ED to give treatment** (medical or psychiatric) against a person's wishes.
- o Consider whether or not the patient has the capacity to refuse treatment. If not, consider whether there is a situation of such urgent necessity that you proceed to treat the patient in their 'best interests' (ie under the common law).
- o Do a brief mental health assessment. Consider whether there are grounds to apply for **involuntary admission** (under the MHA) to a psychiatric unit for treatment of a mental disorder. Seek the advice of a senior colleague and/or contact Psychiatric team.

• WHEN A PATIENT FOLLOWING SELF-HARM ABSCONDS FROM THE ED

- o Telephone the patient and ask him/her to come back for assessment / treatment.
- o Contact the patient's next-of-kin.
- o Contact security to search the hospital area.
- o Consider contacting the Police.
- o Complete an incident form and Inform the relevant clinical team and Document it.

• REFERRAL BY ED STAFF TO PSYCHIATRY

- o All patients following self-harm should be referred to Psychiatry.
- o Please inform the liaison psychiatry team of cases of **suicide** who die in the ED or in the community but are brought to ED by the emergency services.

• REFERRAL TO SOCIAL WORK

- o All patients <18 yrs following self-harm should be referred to the Social Work in addition to Psychiatry.
- o All cases of adult presentation where Child Protection/Welfare concerns are identified.
- o All cases of adult self-harm presentation where Domestic / Elder Abuse is identified

III. ASSESSING SUICIDE RISK

- There are many different risk assessment tools in use. Probably, the most commonly used is the **SAD PERSONS scale**. The accuracy of these scales in predicting future self-harm and suicide is poor.
- The advice from NICE is that a standardised risk assessment scale should only be used to aid identification of those at high risk of repetition of self-harm or suicide, and not to identify those patients who are supposedly 'low risk' who are then not offered services.

FACTORS ASSOCIATED WITH SELF HARM

Demographics	Social isolation
	Lower social class
	Age >45
	Male
	Unemployment
	Single/divorced
	History of violence/criminal convictions
Features in the past medical history	Chronic alcohol and/or drug misuse
	Physical illness
	Previous self-harm
	Psychiatric disorder
	Personality disorder
	History of abuse
Psychological characteristics	Depression
	Hopelessness
	Continued suicidal intent

- Components of the modified **SAD PERSONS scale (DROS=2)**

Sex (Male)	1 point
Age (15-25 or >59 years)	1 point
Depression/hopelessness	2 points
Previous attempt/psychiatric care	1 point
ETOH/Drug abuse	1 point
Rational thinking loss	2 points
Separated/divorced/single	1 point
Organised or serious attempt	2 points
No social support	1 point
Stated future intent	2 points

- This score is then mapped onto a risk assessment scale as follows:
 - 0-5:** may be safe to discharge (depending upon circumstances).
 - 6-8:** probably requires psychiatric consultation.
 - >8:** probably requires hospital admission.

CHAPTER 33. NEEDLESTICK INJURY**OVERVIEW**

- **Approach**

- First aid
- Quantify risk
- Post procedure prophylaxis
- Quality assurance
- Education

MANAGEMENT

- **Stop the procedure**

- Ensure patient and proceduralist is safe
- Take over care if required

- **First aid**

- Express blood from wound
- Wash wound immediately with soap and water (2% chlorhexidine wash)
- Dress

- **Risk stratification**

- Identify source patient and test for HIV, Hep B and C
- Test exposed staff member
- Type injury – Depth, Type, Location, Barriers to transmission (double, single gloved),
- Blood on needle

- **Low risk:**

- Contact with saliva, urines or feces
- Bite with no donor blood
- Blood onto intact skin

- **Moderate risk:**

- Needlestick: solid needle, Hollow needle with no visible blood in hub/syringe
- Small amount of blood onto mucosa or non-intact skin
- Superficial bite with donor blood

- **High Risk:**

- Hollow needle with visible blood
- Deep bite with donor blood on wound
- Large amount of blood on mucosa or non-intact skin

- **Notify patient and family**

- Open disclosure
- Consent for testing

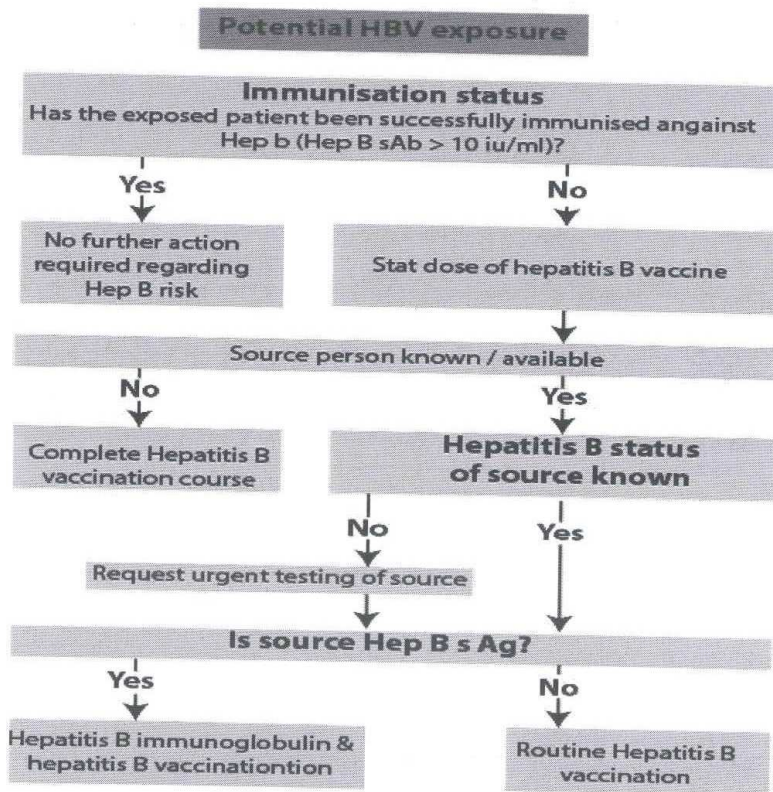
- **Occupational health involvement**

- Initiate the injury reporting system used in workplace (in hours vs out of hours)
- Counselling required with specific risk depending on depth of injury, whether there is visible blood on needle, needle placement in vein or artery, lower risk if solid needle vs hollow
- Document the exposure in detail
- Advice on: safe sex and no blood donation until testing complete

- **Post-exposure prophylaxis**

- Discuss with ID

- **HIV +ve** -> post-exposure prophylaxis within 2 hours
- **Hep B +ve** -> Hep B immunoglobulin
- **Hep C +ve** -> no treatment recommended currently
- **Systems analysis to look at prevention of further events**
 - Document thoroughly
 - Identify factors that may have lead to exposure and could prevent further exposures
 - A unit policy may be appropriate
- **Follow up**
 - Follow up post exposure testing @ six weeks, 3 months and 6 months +/- 1 year
 - If post exposure prophylaxis prescribed -> monitor for toxicity
 - Take precautions (safe sex) to prevent exposing others until follow up testing complete
 - Review of technique with proceduralist



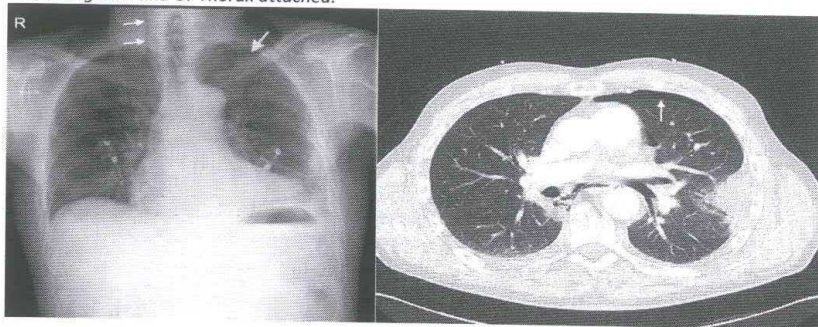
- **Hepatitis B virus prescribing details:**
 - **HBV Vaccine:** Engerix B 1ml IMI (Deltoid) or B Vax II 1ml IMI (Deltoid); will need 2 further injections to complete the course.
 - **HBV Immunoglobulin:** Hepatect CP 0.16-0.2ml/Kg IV infusion at rate of 0.1ml/kg for 10 minutes

CHAPTER 34. VOMITING AND NAUSEA**I. HYPEREMESIS GRAVIDARUM**

- Vomiting is a normal feature of early pregnancy and occurs commonly between 7 and 12 weeks.
- Hyperemesis gravidarum is the presence of intractable, severe nausea and vomiting that results in fluid and electrolyte disturbance, marked ketonuria, nutritional deficiency and weight loss.
- It affects less than 1% of pregnancies.
- **RISK FACTORS FOR HYPEREMESIS GRAVIDARUM**
 - *First pregnancy*
 - *Multiple pregnancy*
 - *Trophoblastic disease*
 - *Obesity*
 - *Prior or family history of hyperemesis gravidarum*
- **POTENTIAL COMPLICATIONS OF HYPEREMESIS GRAVIDARUM**
 - *Renal failure*
 - *Central pontine myelinosis*
 - *Coagulopathy*
 - *Mallory-Weiss tear*
 - *Hypoglycaemia*
 - *Pneumomediastinum*
 - *Rhabdomyolysis*
 - *Wernicke's encephalopathy*
- **ED MANAGEMENT OF HYPEREMESIS GRAVIDARUM**
 - Mild cases of nausea and vomiting in early pregnancy can often be controlled by dietary measures or non-pharmacological measures such as eating ginger and P6 wrist acupressure.
 - In severe cases that are causing heavy ketonuria and marked dehydration admission to hospital is usually required for rehydration with intravenous fluids.
 - **The NICE Clinical Knowledge Summary (NICE CKS)** on nausea and vomiting in pregnancy recommends that if an anti-emetic is required **oral Promethazine** or **oral Cyclizine** should be used first-line.
 - The situation should then be re-assessed after 24 hours. If the response to treatment is inadequate then a second-line drug such as **Metoclopramide**, **Prochlorperazine** or **Ondansetron** should be used.
 - **Metoclopramide** should not be used in patients under the age of 20 due to the increased risk of extra-pyramidal side effects.
 - **Proton pump inhibitors** (e.g. omeprazole) and **histamine H2-receptor antagonists** (e.g. ranitidine) are a useful adjunct in women that also have significant dyspepsia.

II. BOERHAAVE'S SYNDROME

- This 60 year old man presented to ED with acute back pain which had developed after an episode of vomiting. CXR and CT Thorax attached.



Q1. Describe the chest X-ray?

- CXR, left shows a **small left apical pneumothorax** (yellow arrow), as well as some **surgical emphysema** extending along the right side of the neck (white arrows).
- Because of the latter finding, CT was performed and showed a **pneumomediastinum** due to oesophageal perforation (orange arrow). The left pneumothorax is indicated by the white arrow. The patient also has some consolidation in the left lung.

Q2. What is the diagnosis?

- **Boerhaave syndrome** or so-called '**spontaneous**' rupture of the oesophagus.

Q3. What is the classic presentation of this condition?

- "A middle-aged man presenting with sudden-onset severe chest or epigastric pain, often radiating to the back or shoulder, after repeated episodes of retching or vomiting in association with over-indulgence in food and alcohol."
- Most presentations of Boerhaave's syndrome are atypical and the diagnosis often requires a high index of suspicion – usually an "**oesophagram**" of some sort is required.
- In about 1 in 4 cases there is no history of vomiting!

Q4. What is the Mackler triad?

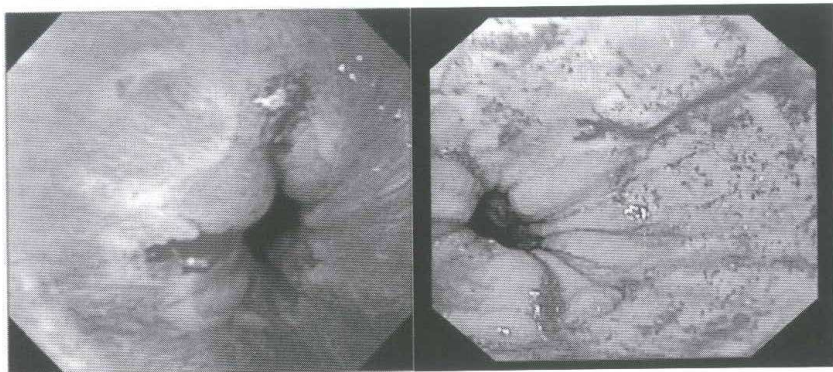
- The **Mackler triad** consists of:
 - Vomiting
 - Lower thoracic pain
 - Subcutaneous emphysema
- Although it supposedly defines the classic features of Boerhaave's syndrome it is probably not worth knowing because it is rarely found and is of negligible clinical utility in the real world.

Q5. Outline the management of this condition.

- This is a highly **lethal condition** – it is essentially 100% fatal if left untreated.
- Overall mortality is about 30%.
- The cornerstones of management are:
 - **Aggressive resuscitation**
 - **Broad-spectrum antibiotics**
 - **Early referral for surgical intervention**

III. MALLORY-WEISS TEAR

- A 21-year-old male presents to the emergency department with hematemesis.
- He had been out drinking every night that week with his friends in celebration of his 21st birthday.
- He reports having vomited each night, but tonight when he started vomiting, his friends noticed that there was streaking of blood and brought him into the emergency department.

**• INTRODUCTION**

- Superficial longitudinal mucosal lacerations of the distal esophagus or proximal stomach
- Associated with forceful retching, alcoholism, and hiatal hernias
- Amount of blood loss is usually small and self-limited
- Accounts for approximately 5% of all presentations of upper GI bleeds

• PRESENTATION

- **Symptoms**
 - Blood in vomit
 - Blood in stool
 - Dark stools
 - Epigastric pain
 - Back pain

• PHYSICAL EXAM

- Upper GI bleed
- Hemodynamic instability
- Can occur with large bleeds
- Signs include hypotension/tachycardia

• EVALUATION

- Mallory-Weiss tears are diagnosed via direct visualization under **endoscopy**

• DIFFERENTIAL

- Esophageal varices, Boerhaave's syndrome, ulcerative diseases of the esophagus (including reflux esophagitis or infectious esophagitis)

ED MANAGEMENT OF MALLORY WEISS TEAR

• Medical management

○ Supportive therapy and observation

- Management of hemodynamic instability including
 - IV fluids
 - Blood transfusion if needed
 - Most bleeds resolve spontaneously
- Refer to Surgery

• Surgical/procedural intervention

○ Upper endoscopy

- First-line treatment for persistent bleeds
- Combined with epinephrine or sclerosant injection, thermal coagulation, banding, or hemoclips to control bleeding

○ Angiotherapy

- Often with left gastric artery embolization
- Performed by interventional radiology
- Indicated in cases refractory to endoscopic treatment
- Surgical repair
- Oversowing of the mucosal tear is rarely indicated even in refractory cases.

• Prognosis

- Bleeding stops spontaneously in 80-90% of patients
- Up to 10% of patients will experience hemodynamic instability
- Recurrence of mallory-weiss tears is rare

• Prevention

- Avoid engaging in activities that lead to excessive coughing or vomiting (i.e. binge drinking).

• Complications

- *Hypovolemic shock,*
- *Organ infarction,*
- *Death if bleeding is not controlled*

CHAPTER 35. WEAKNESS AND PARALYSIS**I. TRANSIENT ISCHAEMIC ATTACK (TIA)**

- A TIA is a brief episode of neurologic dysfunction caused by focal brain or retinal ischaemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of acute infarction.

MAIN CAUSES OF TIA

- TIAs result from one of three main aetiologies:
 - **Large artery atherosclerotic infarction** e.g. carotid artery disease
 - **Small vessel disease** e.g. striate arteries
 - **Embolism from a cardiac source** e.g. left atrial appendage in atrial fibrillation
- Other causes can include:
 - **Dissection**
 - **Hypercoagulable states**
 - **Sickle cell**
- In some patients, the aetiology is undetermined.
- The neurological symptoms and signs will reflect which vascular territory has been affected.

INTRODUCTION TO CEREBRAL CIRCULATION

- The **anterior circulation** is served by the internal carotids, which supply blood to the **anterior 3/5 of the cerebrum**. The main branches of the internal carotids are the:
 - Middle cerebral artery (MCA)
 - Anterior cerebral artery (ACA)
- The **posterior circulation** is served by:
 - The **vertebrobasilar arteries** which supply the **posterior 2/5 of the cerebrum**, part of the cerebellum and the brain stem
 - The basilar artery which gives off the **posterior cerebral arteries (PCA)**
- The anterior and posterior circulations are linked via **posterior communicating arteries** forming the Circle of Willis. The precise area supplied by each artery varies between individuals, as does the presence or absence of collateral vessels.

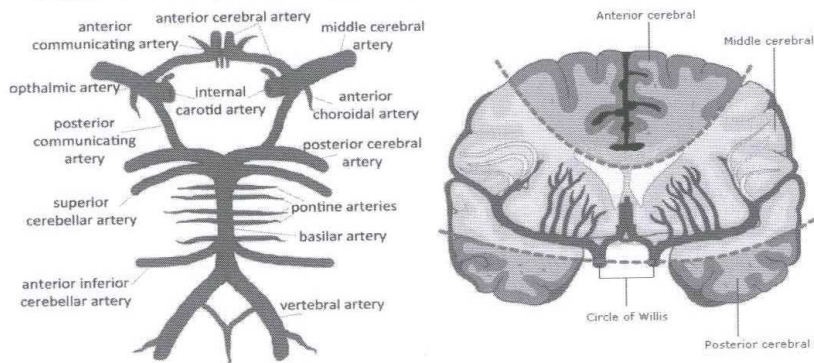
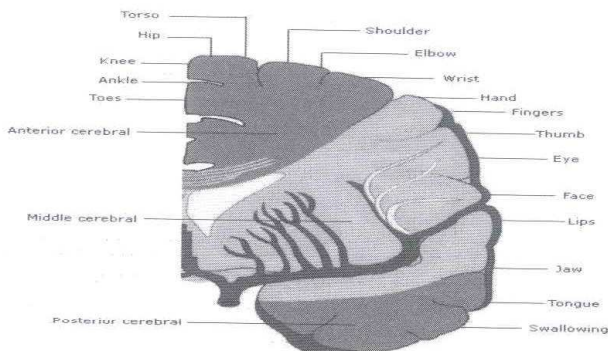


Figure 1.35.1. The circulation regions of the brain.

1. ANTERIOR CEREBRAL CIRCULATION



- The symptoms of TIA in the anterior circulation (Carotid/ACA/MCA) are:
 - **Weakness or sensory loss** affecting the contra-lateral arm, leg or one side of the face.
 - **Dysphasia or dysarthria** (dysphasia usually indicates left sided cerebral hemisphere ischaemia)
 - **Monocular visual loss** (amaurosis fugax) usually lasting a few minutes only (ophthalmic branch of internal carotid)
- In differentiating between anterior and middle cerebral artery occlusions, it should be noted that:
 - **MCA occlusion** affects the contra-lateral face and arm more than the leg.
 - **ACA occlusion** affects the contra-lateral leg more than the face or arm
 - In lesions affecting the **internal capsule** (i.e. small vessel disease), the face, arm and leg may be equally affected as the relevant nerve fibres lie close together.

2. POSTERIOR CEREBRAL CIRCULATION

- The symptoms of TIA in the posterior circulation (vertebrobasilar system, PCA, cerebellum) include:
 - Bilateral motor and/or sensory deficits
 - Cortical blindness
 - Diplopia
 - Vertigo (although not usually in isolation)

CLINICAL ASSESSMENT

- The acute diagnosis of TIA is based on clinical history.
- Witness accounts are useful, but may not be available.
- If a patient still has neurological symptoms when they are seen in the ED, it is more probable that they have had a stroke.
- Important points to establish from the history are:
 - The time of symptom onset and the duration of symptoms
 - Associated symptoms
 - Past medical history of co-morbidities associated with increased risk for TIA and CVA
 - Past medical history of possible causes of neurological deficit other than TIA e.g. malignancy

- In order to differentiate TIA from stroke mimics, it is important to ascertain the following:
 - The symptoms are **acute in onset**
 - Symptoms reach **maximum intensity within seconds**
 - Symptoms **all begin at the same time**
 - There may be **single or multiple episodes**

There are a number of conditions that can be mistaken for a TIA:

- | | |
|-------------------------------|------------------------|
| ◦ Hypoglycaemia | ◦ Migraine |
| ◦ Ocular disorders | ◦ Partial seizure |
| ◦ Peripheral vascular disease | ◦ Vestibular disorders |
| ◦ Arteritis | ◦ Presyncope/syncope |
| ◦ CNS tumour | ◦ Neuropathy |
| ◦ Subdural haematoma | ◦ Radiculopathy |

TIA MANAGEMENT IN THE ED

- When managing patients with TIA in the ED, it is important to consider the aetiology and be able to relate focal neurology to a specific vascular territory.
- Some aetiologies are more amenable to treatment aimed at stroke prevention. The key causes to consider are symptomatic **carotid stenosis** and **atrial fibrillation**.
- Detection of patients with carotid stenosis or atrial fibrillation enables prompt delivery of effective treatment to reduce the probability of a stroke.

ABCD2 Score

- One recent study used a combination of the California score and the ABCD score used in the UK to derive a score designed to predict two-day risk of stroke.
- The derived score ABCD2 has been independently validated in four different groups of patients.

Parameter	Feature	Score
Age	>60	1
Blood pressure	SBP > 140mmHg or DBP > 90mmHg	1
Clinical features	Unilateral weakness	2
	Speech impairment with no weakness	1
	Other	0
Duration of symptoms	>60 minutes	2
	10-59 minutes	1
	<10 minutes	0
Diabetes	Yes	1
0-3: low risk		4-6: Moderate Risk
		6-9: High Risk

- Patients with a **score of 4 or more** should receive:
 - *Immediate aspirin (300mg),*
 - *Specialist assessment **within 24 hours of symptom onset** and*
 - *Secondary prevention as soon as the diagnosis is confirmed.*
- **Low-risk patients** should receive the same care **but within 7 days of symptom onset**.
- Other **high risk patients** are:
 - *Those with new onset atrial fibrillation (AF)*
 - *Patients already on warfarin.*
 - *Those who have had more than one TIA in a week.*

INVESTIGATIONS

- Standard investigations should include:
 - **Blood tests:** Plasma glucose FBC, U&E, Lipid profile, LFTs
 - **ECG**
 - **Brain imaging** (NICE: **MRI with diffusion weighting** within 24hrs of onset is the gold standard)
 - **Carotid Doppler**

1. CAROTID STENOSIS

- While a bruit (an abnormal sound heard over a blood vessel) is suggestive of carotid disease, it is not possible to determine who would benefit from carotid endarterectomy from clinical examination alone.
- The value of carotid surgery is directly linked to the severity of the carotid stenosis and decreases with time.
- Imaging should be done as soon as possible to enable urgent surgery, because the greatest benefit is in the first two weeks after the event.

MANAGEMENT OF CAROTID STENOSIS

- Initiating **antiplatelet therapy**, a **statin** and **antihypertensive** treatment with **urgent carotid imaging** and **surgical referral as needed**, is associated with an 80% reduction in the risk of early recurrent stroke.
- **Antiplatelet therapy**
 - Start **aspirin before imaging** in patients with suspected TIA.
 - If TIA occurs while already on low-dose aspirin, increasing the dose will not help; consider adding **dipyridamole** and **arrange specialist assessment**.
 - In patients who cannot take aspirin, **clopidogrel** or **dipyridamole** can be used.

2. PATIENTS IN AF

- A patient who has had a TIA and is in atrial fibrillation is at increased risk of further TIAs and stroke.
- A full assessment of their stroke risk should be undertaken.
- Most will benefit from **anticoagulation: Warfarin** reduces the risk of recurrent stroke.

DISPOSAL

- **low risk:** Refer to local TIA service
- **Moderate- and High-risk:** immediate access to thrombolytic therapy is available.

Management Principles

- What are the principles underpinning the management of suspected TIA?
 - Exclusion of other serious causes for the symptoms
 - Antiplatelet therapy
 - Identification of carotid stenosis
 - Identification of cardiac disease such as AF
 - Modification of risk factors such as smoking, hypertension, hyperglycaemia and high cholesterol

II. STROKE IN THE ED

INTRODUCTION

- A suspected stroke should be treated as a medical emergency.
- **An acute stroke** is the clinical result of an interruption of the blood supply to a focal part of the brain causing loss of neurological function.
- Approximately **85%** of strokes are caused by occlusion of one of the arteries supplying the brain (**ischaemic stroke**) and approximately **15%** are caused by non-traumatic intracerebral haemorrhage (ICH). (We will just talk about Ischaemic strokes)
- **Circulation Territories:** refer to TIA

CLINICAL ASSESSMENT AND RISK STRATIFICATION

1. FAST SCORE

- A useful tool for pre-hospital assessment of suspected acute stroke is FAST: **Face – Arm – Speech – Test**. There are public awareness campaigns using FAST (Stroke – Act FAST), and patients at high risk of stroke should be given information about calling for immediate help at the onset of symptoms.
- **FAST: Score 1 point each feature: Maximum score 3, Minimum score 0**
 - Face weakness
 - Arm weakness
 - Speech disturbance
- FAST is used by the ambulance services, and this is now a component of paramedic training and documentation in the UK. It is also part of the pre-alert information given to the hospital prior to the patient's arrival.

ED PROCEDURES

- In the ED, the initial priorities are a **rapid structured assessment**, and **exclusion of hypoglycaemia**.
- Recognition Of Stroke In the Emergency Room: **ROSIER**: score from -2 to +5

2. ROSIER SCORE

Clinical Hx: Score -1 for each	Neuro signs: Score +1 for each "FALS V"
<ul style="list-style-type: none"> • Loss of consciousness • Convulsive fit 	<ul style="list-style-type: none"> • Face weakness • Arm weakness • Leg weakness • Speech disturbance • Visual field defect
<ul style="list-style-type: none"> • A score of 1 or above: makes stroke more likely If the score is negative: another diagnosis should be considered: a stroke mimic. 	

INVESTIGATIONS

1. Non-Contrast CT

- The main aim is to exclude a bleed as the cause of the focal neurological signs.
- It may also show another cause, such as a brain tumour or subdural haemorrhage.
- Early signs of ischaemia may be seen, for example the loss of differentiation of the grey and white matter interface.

- The **National Clinical Guidelines for Stroke** recommend that brain imaging should be performed **immediately** (ideally the next appointment slot, and definitely **within one hour**) for people with acute stroke who have:
 - **Contra-indications for thrombolysis or early anticoagulation treatment**
Or are:
 - On anticoagulant treatment**Or have:**
 - A known bleeding tendency
 - A depressed level of consciousness
 - Glasgow coma score (GCS) below 13
 - Unexplained progressive or fluctuating symptoms
 - Papilloedema, neck stiffness or fever
 - Severe headache at onset of stroke symptoms
- The guidance goes on to say: "**For all people with acute stroke without indications for immediate brain imaging, scanning should be performed as soon as possible.**"

2. MRI

- MRI has a higher sensitivity for early acute ischaemic changes and can image the posterior fossa more reliably. With **diffusion-weighted MRI**, diffusion perfusion mismatch may show the area of potentially reversible ischaemia, and better identify patients, beyond the current guidelines, who would benefit from thrombolytic therapy.

3. Other Tests

All patients

- | | |
|--------------------|-------|
| ◦ FBC | ◦ ESR |
| ◦ U&E | ◦ ECG |
| ◦ Lipid profile | ◦ CXR |
| ◦ Clotting profile | |

Selected patients:

- Toxicology screen
- Pregnancy test
- LFTs

ED MANAGEMENT OF STROKE

- **Early CT scan**
 - Ideally **within 1 hour ED arrival**, if any of: indications for lysis or early anticoagulation; on warfarin; known bleeding tendency; depressed GCS <13; unexplained progressive or fluctuating symptoms; suspected meningitis; severe headache at onset.
 - Otherwise **within 24 hours** (see later).
- **General supportive** (see under Stroke Units / Teams below)
 - Airway protection, correction of hypoxia, treatment of hypoglycaemia or hyperglycaemia, treatment of infection, maintenance of normothermia, avoidance of aspiration, and pressure area care if unconscious.
 - Consider BP control, though no agent significantly affects outcome.
 - Calcium channel blockers (nimodipine / nicardipine), ACEI, and GTN all have been used. Note thrombolysis requires SBP < 185/ DBP < 110 mmHg to qualify.
 - Avoidance of hypotension equally important.
- **Antiplatelet treatment:**
 - **Aspirin 300 mg orally** (or via NGT / PR) **early and then daily** if stroke is non-haemorrhagic on CT or for TIA, ideally within first 24 hours. Continue for at least 2 weeks.

- Addition of **dipyridamole 200 mg po bd** for TIAs and minor ischaemic stroke preferred, but note more side effects including headache!
- **Anticoagulant treatment:**
 - No net advantages of heparin over antiplatelet agents.
 - No clear advantages of LMWH / heparinoids over UFH. Need more data.
 - LMW or UF heparin reduce DVT/PE, but not stroke deaths or dependency.
 - Glycoprotein IIb/IIIa antagonists have an unknown effect.
- **Thrombolysis:**
 - **The indications for considering thrombolysis are:**
 - *Clinical signs and symptoms consistent with acute stroke*
 - *Clear time of onset*
 - *Presentation within 4.5 hours of onset*
 - *No contra-indications*
 - **The recommended medication is:**
 - **Alteplase at 0.9mg/kg body weight (maximum dose 90mg)** given as an infusion over 60 minutes, with the first 10% of the total dose administered as a bolus.

CONTRA-INDICATIONS FOR THROMBOLYSIS OF ACUTE ISCHAEMIC STROKE

- **Contra-indications found on brain imaging include:**
 - Haemorrhage
 - Greater than one-third MCA territory acute ischaemic change
 - Extensive small vessel disease
- **Contra-indications from history include:**
 - History of stroke or head injury in the last 3 months
 - Major surgery or trauma in the last 14 days
 - History consistent with subarachnoid haemorrhage
 - History of previous intracranial haemorrhage
 - History of seizure at stroke onset
 - History of gastrointestinal or urinary tract haemorrhage within 21 days
 - Recent arterial puncture at a non-compressible site
 - Recent lumbar puncture
 - Heparin treatment within last two days
- **Contra-indications from investigations include:**
 - Haemoglobin <10g/dl
 - Platelets <100x10⁹/l
 - INR >1.7
 - Glucose <2.7mmol/l
- **Contra-indications from examination include:**
 - Symptoms rapidly improving
 - Low NIHSS score (4 or less) or very high NIHSS score (25 or more)
 - Systolic blood pressure consistently >185mmHg
 - Diastolic blood pressure consistently >110mmHg
- **Time Benchmarks for Potential Thrombolysis**
 - *Door to CT scan completion: 25 minutes*
 - *Door to CT scan interpretation: 45 minutes*
 - *Door to treatment: 60 minutes*

III. FACIAL NERVE PALSY

1. BELL'S PALSY

INTRODUCTION

- Isolated facial muscle weakness is an uncommon presentation to the ED and may be quickly diagnosed by the unwary as Bell's palsy.
- The Emergency Physician must be aware of two potential pitfalls when presented with a patient with facial weakness:
 - *Central facial weakness must be differentiated from a peripheral palsy*
 - A diagnosis of Bell's palsy should only be made after exclusion of other causes for a peripheral facial muscle weakness.
- *Bell's palsy is defined as an acute idiopathic **peripheral facial nerve paresis** and is the most common cause of acute peripheral facial weakness.*
- 84% of patients with Bell's palsy will recover full or near normal function, without any treatment.
- **COMMON SEQUELAE FOUND IN THOSE THAT FAIL TO RECOVER**
 - Residual partial facial weakness
 - Facial contracture
 - Ageusia: loss of taste function of the tongue
 - **Motor synkinesis:** involuntary muscle movement accompanying a voluntary movement e.g. eye closure when smiling
 - **Autonomic synkinesis:** e.g. Crocodile tears syndrome when lacrimation occurs with salivation
 - Hearing loss

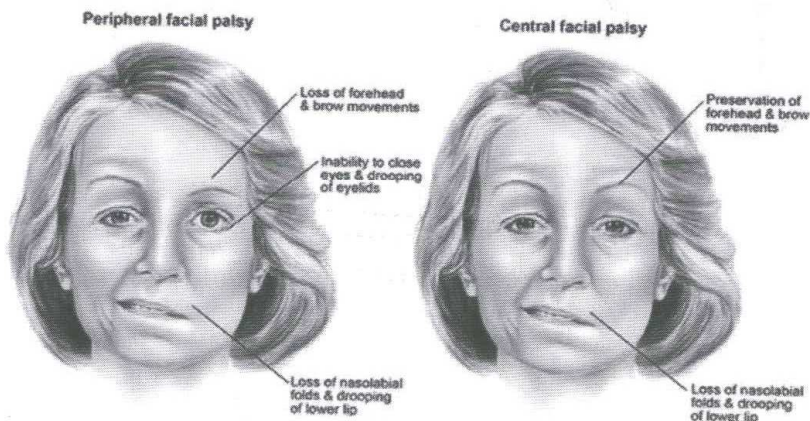


Figure 1.35.2. Peripheral and central facial palsy

PATHOPHYSIOLOGY

- The cause of Bell's palsy has long been debated but only recently has evidence started to accumulate for a viral origin.
- **Reactivation of latent herpes simplex or zoster virus** is the most likely scenario.

CLINICAL ASSESSMENT

- **History**

- o Unilateral facial weakness noticed by the patient themselves or a family member.
- o Approximately 50% of patients with Bell's palsy experience facial pain, aural fullness or postauricular pain and in 25% of cases this precedes the paresis by 2-3 days
- o Numbness or altered sensation to the same side of the face are also often described although formal testing should reveal normal sensory function.
- o Other symptoms such as fever, headache or other neurological or systemic upset.
- o Other symptoms which are commonly found in Bell's palsy are attributable to individual branches of the facial nerve.

- **Examination**

- o Two questions must be asked by the clinician when assessing a patient who presents with an acute facial weakness:
 - *Is this an upper or lower motor neurone lesion?*
 - *Is this an idiopathic peripheral facial muscle weakness (Bell's palsy) or is there another cause for the problem?*

- 'Bell's phenomenon', is the rolling upwards and outwards of the eye on the affected side when attempting to close the eye and bare his teeth.

1. **Is this an upper or lower motor neurone lesion?**

- The key to differentiating a central (upper motor neurone) from a peripheral (lower motor neurone) facial palsy is to **identify the extent of facial muscle weakness**.
- The muscles of the **upper half of the face** (frontalis, corrugator and orbicularis) are innervated bilaterally by corticobulbar fibres.
- **Weakness of the forehead muscles indicates a peripheral facial nerve problem whereas sparing of the forehead muscles is diagnostic of a central lesion.**
- Asking the patient to close their eyes tightly or wrinkle their forehead will quickly identify the source of the problem. A patient with facial weakness who cannot close their eye tightly or raise their eyebrow has a peripheral facial palsy.

2. **Is this an idiopathic peripheral facial muscle weakness (Bells palsy) or is there another cause for the problem?**

- Having identified a peripheral facial muscle weakness, the clinician must then exclude other causes before diagnosing an idiopathic palsy (Bells palsy).
- General examination of the patient should be normal in Bells palsy.
- Derangement of vital signs, fever, rash or other abnormality on examination all point to an alternative cause for facial weakness such as:
 - o *Lyme disease/ Sarcoidosis/ HIV infection/ Diabetes*
 - o *Ramsey-Hunt syndrome (Herpes zoster virus)*

In addition, acute peripheral facial weakness may either be found in known diabetics or be a presenting feature of previously undiagnosed diabetes.

- **Other causes of an isolated lower motor neurone facial nerve palsy include:**

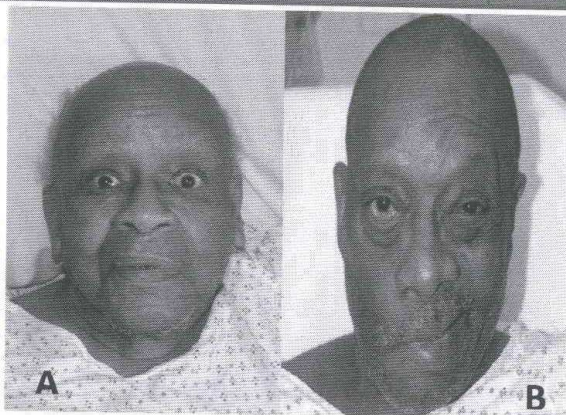
- o Trauma, Parotid gland tumour
- o Cerebello-pontine angle tumour e.g. acoustic neuroma
- o Middle ear infection, Cholesteatoma

INVESTIGATIONS

- As Bells palsy is an idiopathic condition, there is no currently available diagnostic test which will confirm the diagnosis. The only other investigations required are those to exclude a secondary cause for facial weakness.

Bell's Palsy Vs STROKE

- **Patient A** shows a flattened nasolabial fold and inability to smile on the affected side with sparing of the forehead and eye closure muscles and resulting in a partial paralysis of the face which is caused by a **Stroke**.
- **Patient B** shows flattening of the nasoabial fold, widened palpebral fissure, and absence of forehead wrinkles on the right. This lesion is what causes **Bell's palsy**.



HOUSE-BRACKMANN CLASSIFICATION

GRADE	DESCRIPTION	CHARACTERISTICS
I	Normal	Normal facial function
II	Mild dysfunction	Slight weakness noticeable on close inspection. May have slight synkinesis
III	Moderate dysfunction	Obvious but not disfiguring difference between two sides. Complete eye closure with effort. Noticeable but not severe synkinesis, contracture or hemifacial spasm
IV	Moderately severe dysfunction	Obvious weakness or disfiguring asymmetry. Normal symmetry or tone at rest. Incomplete eye closure.
V	Severe dysfunction	Only barely perceptible motion. Asymmetry at rest.
VI	Total paralysis	No movement

ED MANAGEMENT OF BELL'S PALSY

- Management of Bells palsy can be divided into:
 - Treatment directed at the facial nerve
 - Treatment of the consequences of the facial nerve palsy
- **Treatment directed at the facial nerve**
 - **Prednisolone 50 or 60mg for a total of 10 days**, one of the studies tapering the dose after 5 days.
 - The addition of an antiviral agent to steroid treatment in Bells palsy has not been shown to provide any additional benefit
- **Treatment of the consequences of facial muscle weakness.**
 - The most common problem associated with facial muscle weakness is **incomplete closure of the eyelid**.
 - This may lead to **exposure keratitis** and **corneal ulcers**.
 - **Hourly lubricating eye drops** during the day and **eye ointment** at night
 - **Eye taping at night**.
 - **Referred for ophthalmology** follow-up as further options include temporary tarsorrhaphy or botulinum toxin tarsorrhaphy are available.
 - Attempts have been made to reduce facial muscle weakness by **physiotherapy**, **electrical stimulation** and **acupuncture**.

2. RAMSAY HUNT SYNDROME

- Ramsay Hunt syndrome (also termed **Hunt's Syndrome** and **Herpes zoster oticus**) is a rare neurological disorder characterized by **paralysis of the facial nerve** (facial palsy) and a **rash affecting the ear or mouth**.
- It is caused by **reactivation of herpes zoster virus** that has previously caused chicken pox in the patient.

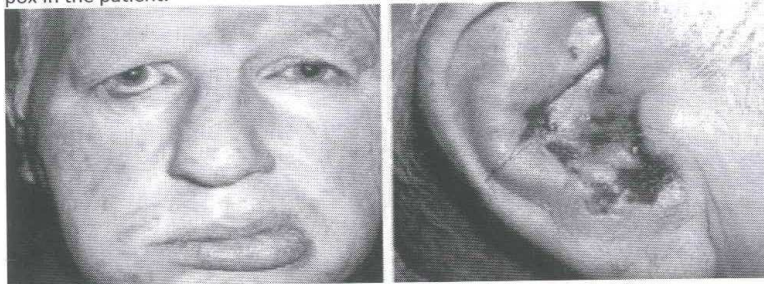


Figure 1.35.3. Ramsay Hunt Syndrome with a vesicular rash in the left ear

- It is important to note that the rash may be initially erythematous only, with vesicles developing later.
- Ramsay Hunt syndrome is commonly accompanied by associated symptoms such as **hearing loss and vestibular disturbance** due to involvement of structures adjacent to the facial nerve.
- It is associated with a poorer prognosis than Bells palsy and sequelae such as **persistent synkinesis and hearing loss**, are more common.
- Rx: **Prednisolone 60mg once daily for 10 days and acyclovir 800mg five times a day for 7 days** are associated with improved rates of recovery.
- Other treatment and follow-up for the sequelae of facial muscle weakness are also be required, as for Bells palsy.
- In addition the patient must be counselled on the infectivity of the rash and potential risk to the non-immune and immunocompromised.

3. ZOSTER SINE HERPETE

- It is a term for Ramsay Hunt syndrome without apparent vesicles or when there is a delayed presentation of the rash.
- In 15% of patients with Ramsay Hunt syndrome vesicles develop after the onset of facial weakness and therefore patients diagnosed with Bells palsy must be instructed to return or see their GP should a rash develop later.

CHAPTER 36. WOUND MANAGEMENT

I. GENERAL APPROACH

• History

- Time it occurred
- Mechanism of injury
- Possibility of foreign body
- Loss of function of structures beneath
- **General factors**
 - A history of diabetes
 - Steroid therapy, Peripheral vascular disease
 - Ask about tetanus status
- Any wound on the hand must lead to enquiry about profession and whether the patient is self-employed.

• Examination

- Examine to detect INDIVIDUAL structures that could be damaged (e.g. tendons, nerves), for the presence of dirt, foreign bodies and the displacement and loss of tissue. (e.g. use "DP" or "FDS" not "tendons" intact)
- Check the skin edge of viability.
- If a skin flap has been raised record the dimension in terms of width, length and orientation of the base of the flap.
- Make an accurate record of your clinical findings.
- All Wounds caused by glass must always be x-rayed.

• Treatment

- Thorough mechanical cleaning is essential for all wounds, e.g. for dirty hands get the patient to use tap water, Hibiscrub himself or Swarfega if grease is present.
- Remember to use scrubbing/toothbrushes if necessary. Local anaesthesia will probably be required to assess and clean the wound thoroughly.
- Wounds may be closed by:
 - **Primary suture:** for clean wounds less than 6 hours old and for clean incised wounds that can be closed tension free.
 - **Delayed primary suture:** 3-4 days for wounds that are potentially infected - daily dressings required
- Wounds should not be closed if they are dirty, old, if there is a possibility of a foreign body, crush injury, cannot be closed without tension or are due to a bite (except on the face) - use DELAYED primary closure. Clean and dress the wound and review it at 48 hours. If it is not infected, then close it between days 2 and 5.

• Suturing

Wound	Suture	Removal days
Scalp	3/0 4/0	7
Face	5/0 6/0	4 - 5
Anterior trunk	4/0	7 - 10
Posterior trunk	3/0	7 - 10
Upper limbs	4/0	7 - 10
Hands	5/0	7
Lower limbs	3/0	10 - 14
Extensor surface joints		14

- o The wound should be sutured so that at the end it is completely closed throughout its depth and length.
- o **Avoid any dead space.** (Achieved with vertical mattress stitches without tension) with 5/0 Vicryl.
- o **Interrupted suture** should always be used.
- o The knots should be placed to one or other side of the wound and must not be tied tightly to allow for swelling.
- o Knots should be placed at least **2 mm from the skin edge and 3 mm apart** (hand).
- o All suturing is the responsibility of the SHO / ENP treating the patient.
- o When medical students or dental students suture, the assessment of the wound and suggestion for suturing must be made by the SHO / ENP who will also need to check the wound after suturing.
- o Remember sutures on extensor surfaces of joints need to stay in longer and the joint may need immobilisation to produce a good scar.
- o **Record the number of sutures** inserted as this helps nurses/patients when they remove them. If the patient is referred back to the GP's Practice Nurse for removal of sutures, the number, type and date of removal must be indicated in the GP letter given to the patient.

1. INFECTED WOUNDS

- o Do NOT suture closed.
- o Remember the importance of immobilisation and elevation in the treatment of sepsis, e.g. high sling for hands.
- o Take a swab to identify the organism in every case.
- **Wound packing**
 - o Wound cavities are not to be packed as this maintains a cavity, traps infection, increases scarring and slows healing.
 - o The aperture is kept open by means of a small plastic corrugated drain or wick to allow the cavity to heal in and simultaneously discharge unhealthy material.
 - o Alternatively, an elliptical incision will keep the aperture open.
 - o Please drain rather than pack.

2. BITES (ANIMAL AND HUMAN)

- o Infection is a very real risk
- o The mouth harbours many organisms
- o The wounds have contused tissues in addition to the lacerations
- o Therefore, thorough cleaning often using irrigation with H₂O₂ (Hydrogen peroxide)
- o Excision of damaged tissues is often necessary
- o **Do not close except in facial wounds**
- o Delayed primary suturing prevents wound complications
- o Large wounds can be partially closed, particularly the subcutaneous tissues.
- o Antibiotics are indicated for human bites and all bites to the hand.
- o Initially **Co-amoxiclav** is usually sufficient BUT swab all bites and review the patient in 2 days.
- o The bacteriology result will then be available to guide you for more antibiotics if necessary.
- o Remember - rest and elevation.

3. ABSCESSES

- o An abscess is a contained infection which is treated surgically.
- o Antibiotics are only required if there is cellulitis or lymphadenopathy spreading from the focus of infection.

4. CELLULITIS

- o Cellulitis is usually caused by streptococci or staphylococci.
- o Treatment is with **Penicillin and Flucloxacillin together (500 mg QDS)**.
- o On presentation mark the area of cellulitis, look for lymphangitis, lymphadenitis and pyrexia. The presence of these features or spreading cellulitis require referral and hospital admission.
- o Check and record BM (Beware of diabetic or immunocompromised patients).
- o If the area is small, the patient is sent away with a course of antibiotic and reviewed at 24 hours.
- o If the cellulitis has increased that is an indication for admission for elevation of the affected part and intravenous antibiotics.
- o Diabetic patients who are well but have a small area of cellulitis should be treated with **Ciprofloxacin** and reviewed early.

• ANTIBIOTIC POLICY

- o Do not give prophylactic antibiotics
- o Thorough cleaning and debridement of the wound is needed
- o **Co-amoxiclav** is only recommended for bites and penetrating wounds (e.g. after standing on a nail) or after appropriate sensitivity tests from the microbiology department
- o **Ampicillin and Flucloxacillin** are recommended for cellulitis
- o **Incision and drainage** is the treatment of choice when pus is present.

• CONDITIONS REQUIRING ANTIBIOTIC TREATMENT:

- o Human or Animal Bites (rarely needed in head / neck wounds)
- o Penetrating lacerations overlying joints or fractures
- o Lacerations of the palm or sole (note no conclusive evidence for penetrating wounds to the sole)
- o Contaminated wounds
- o Cellulitis / lymphangitis
- o Wounds in Insulin-dependent diabetics or those on steroids or immunosuppressed

II. BITES WOUNDS

BACKGROUND

- **Dog bites** are the most common bite injury (account for 80-90% of presentations).
- **Cats bites** become more frequently infected than dogs.
- **Human bite** wounds account for 2-3% of bite presentations.
- **Clenched fist injuries**
 - Are the most severe of human bite injuries.
 - Commonly present as a small wound over the MCPJ of the dominant hand (patient striking another person's teeth)
 - Human bite wounds to the hand more commonly develop bacterial infection than human bites at other sites, with clenched fist injuries conferring the highest risk, particularly because of the potential for breaching the MCP joint space to produce **septic arthritis** or **osteomyelitis**.
 - Clinical examination should focus on the possibility of **extensor tendon injury** and **joint penetration**.
 - Extensor tendon retracts when the hand is opened, so evaluation needs to be done with the hand in both the open and the clenched positions.

ED MANAGEMENT OF BITES WOUNDS

- Intact skin surrounding dirty wounds can be scrubbed with a sponge and 1% iodine solution.
- **Copious irrigation** (warmed solution 33-37°C) of the wound with normal saline using a **19-G syringe** is necessary.
- Wounds that are dirty and contain **devitalized tissue** should be cleaned with gauze and **debrided**.
- Fresh head and neck wounds can generally be primarily closed.
- **Bite wounds to the hand or feet should be left open** for delayed primary closure or secondary intention. Non-puncture wounds elsewhere may be safely treated by primary closure after thorough cleaning.
- Complete management of bite injuries should include consideration of **tetanus immunisation**.
- For potential hepatitis exposure cases, please see needlestick section.

PROPHYLACTIC ANTIBIOTICS

- Use of oral antibiotics for all types of dog bite wounds reduces the risk of infection by nearly half. Prophylaxis is generally given for 5-7 days.
- **Dog and Cat bites**
 - Use **Co-Amoxycylav**.
 - In penicillin allergy use **Erythromycin** or **clindamycin** plus **Ciprofloxacin**, or **clindamycin** plus **trimethoprim-sulfamethoxazole**.
- **Human bites**
 - Use **Co-Amoxycylav**.
 - In penicillin allergy use **clindamycin** plus either **Ciprofloxacin** or **trimethoprim/sulfamethoxazole** or **doxycycline** (to treat *Eikenella corrodens*).
- Wounds of low risk (face, scalp, ears or mouth, large, clean lacerations) should be re-evaluated in **2 days' time**.
- High risk (all other parts of the body, puncture wounds, immunocompromised patients) should be re-evaluated in **1-day time**.

III. TETANUS

- Tetanus is a notifiable disease and infections are now rare in the UK.
- *Clostridium tetani* produces an exotoxin that blocks inhibitory neurons in the CNS.
- Tetanus spores are present in soil or manure and may be introduced into the body through a puncture wound, burn, or scratch.
- The bacteria grow anaerobically at the site of the injury and have an incubation period of **4–21 days**.
- The disease is characterized by generalized rigidity and spasm of skeletal muscle.
- The muscle stiffness usually involves the jaw (lockjaw) and neck, and then becomes generalized.
- In severe cases, muscle spasms affect breathing and swallowing.
- Autonomic disturbance causes profuse sweating, tachycardia, and hypertension, alternating with bradycardia and hypotension.
- The vaccine is a cell-free toxin extract from a strain of *C.tetani*.
- The immunization schedule for tetanus involves five doses of vaccine at appropriate age intervals.

TETANUS IMMUNIZATION SCHEDULE

- **Primary immunizations**
- The primary tetanus immunizations are given with Diphtheria, pertussis, polio, and Hib vaccines at the following intervals:
 - 2 months old.
 - 3 months old.
 - 4 months old.
- **Reinforcing immunizations**
- Tetanus boosters are combined with diphtheria, pertussis, and polio vaccines. The timing of the boosters is as follows:
 - 1st booster—between ages 3½ to 5 years (ideally 3 years after completion of primary course).
 - 2nd booster—between ages 13 and 18 years (ideally 10 years after the 1st booster).

TETANUS-PRONE WOUNDS

- **Tetanus-prone wounds include:**
 - Wounds or burns that require surgical intervention that is delayed for more than 6 hours.
 - Wounds or burns that show a significant degree of devitalized tissue or a puncture-type injury, particularly where there has been contact with soil or manure.
 - Wounds containing foreign bodies.
 - Compound fractures.
 - Wounds or burns in patients who have systemic sepsis.
- High-risk tetanus-prone wounds are those heavily contaminated with material likely to contain tetanus spores and/or extensive devitalized tissue.
- If the patient has a **high-risk tetanus-prone wound**, they should receive **human tetanus immunoglobulin**, regardless of their immunization history.
- Tetanus vaccine given at the time of a tetanus-prone injury may not boost immunity early enough to give additional protection within the incubation period of tetanus.

- Therefore, tetanus vaccine is not considered adequate for treating a tetanus-prone wound.
- However, this provides an opportunity to ensure that the individual is protected against future exposure.
- Patients who are immunosuppressed may not be adequately protected against tetanus, despite having been fully immunized.
- They should be managed as if they were incompletely immunized.
- For those whose immunization status is uncertain, and individuals born before 1961 who may not have been immunized in infancy, a full course of immunization is likely to be required.
- Injecting drug-users may be at risk from tetanus-contaminated illicit drugs, especially when they have sites of focal infection, such as skin abscesses, that may promote growth of anaerobic organisms.
- Every opportunity should be taken to ensure that they are fully protected against tetanus.
- Booster doses should be given if there is any doubt about their immunization status.
- **Dosage of human tetanus immunoglobulin**
 - The dose of **Tetanus immunoglobulin** is **250 IU IM**, or **500 IU** if more than 24 hours have elapsed since the injury; or there is a risk of heavy contamination; or following burns.
 - The immunoglobulin injection must be given at a different site to the tetanus booster.
- **ED MANAGEMENT OF TETANUS INFECTION**
 - **Supportive:** Paralysis and intubation may be required if breathing becomes inadequate.
 - **Diazepam:** to control muscle spasms.
 - **Wounds:** cleaned and debrided.
 - **Broad-spectrum antibiotic cover.**
 - **Human tetanus immunoglobulin:** 5000–10,000 IU as an intravenous infusion.

ANAESTHETIC COMPETENCES

4 QUESTIONS

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CHAPTER 1. PREOPERATIVE ASSESSMENT

I. HISTORY

- It is important to take a thorough patient history, which includes the following components:
 - **History of Present Illness** (HPI: the concise history behind the current medical condition leading up to the surgical intervention and what is the specific surgical intervention),
 - **Past Medical History** (PMH: important to document comorbidities, especially those involving heart, lungs, liver, and kidneys, paying particular attention to hypertension, diabetes, coronary artery disease, reactive airway disease, recent pulmonary infections, and history of stroke or myocardial infarction),
 - **Past Surgical History** (PSH: type of surgery, type of anesthesia received (regional, monitored anesthetic care (MAC), or general anesthesia),
 - **Any complications from anesthesia** (difficult intubation, prolonged wake up, difficulty with ventilation, post-operative nausea and vomiting, etc.), and any prior anesthetic records available for the medical chart),
 - **Allergies** (including specific reaction), current medications (particular attention to the dosages and last administration of blood pressure medications, anti-platelet medications (Aspirin, Plavix), diabetic medications (oral and parenteral) and steroids),
 - **Family Medical History** (family member having problems with general anesthesia),
 - **Social History** (tobacco use, alcohol consumption, and illicit drugs).

II. CLINICAL EXAMINATION

Physical Exam

- **General**
 - BMI
 - Jaundice
- **Airway**
 - Mallampati score/Mouth opening
 - Cervical spine mobility
 - Temporomandibular joint mobility
 - Teeth (especially diseased/loose/artificial)
 - Thyromental distance
- **Pulmonary**
 - Auscultation
 - Thoracic shape and expansion
 - Oxygen saturation on room air
- **CV system**
 - Auscultation for murmurs
 - Pulses/ BP (including postural drop if relevant)/heart rate/rhythm
 - Venous access sites
 - Edema
 - Venous pressure
- **CNS**
 - Motor & sensory function
 - Cognitive function

- **Hematologic**
 - Petechiae
 - Bruising
 - Clinical evidence of anemia

Common Home Medications Which Affect Anesthesia

Drug	Effect
ETOH	Tolerance to anesthesia
B-blockers	Bronchospasm
Antibiotics	Prolongation of NMJ blockade
Benzodiazepines	Tolerance to anesthesia
Diuretics	Hypovolemia, hypokalemia

III. SPECIFIC ANAESTHETIC EVALUATION

- **Routine Labs and Testing**
 - Patients who are in optimal living conditions and who are undergoing a procedure with minimal risks do not need preoperative labs.
- **ECG**
 - In the past ECG has been recommended at 40 years of age for men for all general anesthetics (women at 50).
 - *Patients at higher risk of having significantly abnormal ECGs which would potentially affect management were those older than 65 yr of age or who had a history of heart failure, high cholesterol, angina, myocardial infarction, or severe valvular disease.*
- The United Kingdom National Institute for Health and Clinical Excellence (NICE) issued a guideline on this topic in 2003 based on a systematic review of the literature.
- In addition to the issue of routine testing, current attempts to improve cardiac outcomes may have led to excessive use of invasive cardiac evaluation.
- Thus, "These results confirm that most patients, even high-risk patients who are undergoing high-risk surgery, will have similar outcomes regardless of whether or not they undergo invasive procedures beforehand.
- Such procedures should be performed only in patients with unstable ischemic coronary disease.

Age	General Anesthesia	MAC/Regional Anesthesia	Nerve Block
< 40	Female: Hb/Hct? Preg. test?	None	None
40 – 49	Male: ECG, Female: Hb/Hct? Preg. test?	None	None
50-64	Hb or Hct, ECG	Hb or Hct	None
65-74	Hb or Hct, ECG, BUN/Creat, Gluc	Hb or Hct, ECG	Hb or Hct
> 74	Hb+Hct, ECG, BUN/Creat, Gluc, CXR?	Hb+Hct, ECG, BUN/Creat, Gluc	Hb+Hct, ECG

- **Overall Risks**
 - 0.2% risk of death within 48 hours for ALL operations, due to anesthesia in 0.01% of procedures.
 - Emergency and vascular surgery are associated with increased risk as are procedures with large blood loss or fluid shifts, with risk of death approaching 5% for certain procedures.
 - ASA status, for all its shortcomings, does well as a predictor of outcome.

ASA CLASSIFICATION

ASA	Health status of patient
I	A normal healthy patient
II	A patient with mild systemic disease
III	A patient with severe systemic disease
IV	A patient with severe systemic disease that is a constant threat to life
V	A moribund patient who is not expected to survive without the operation
VI	A declared brain-dead patient whose organs are being removed for donor purposes

INFORMING THE PATIENT AND CONSENT

- **What is consent?**
 - It is an agreement by the patient to undergo a specific procedure.
 - Only the patient can make the decision to undergo the procedure, even though the doctor will advise on what is required.
 - Although the need for consent is usually thought of in terms of surgery, in fact it is required for any breach of a patient's personal integrity, including examination, performing investigations and administering an anaesthetic.
 - A patient can refuse treatment or choose a less than optimal option from a range offered (providing an appropriate explanation has been given — see below), but he or she cannot insist on treatment that is not on offer.
- **What about an unconscious patient?**
 - This usually arises in the emergency situation, for example a patient with a severe head injury.
 - *Asking a relative or other individual to sign a consent form for surgery on the patient's behalf is not appropriate, as no one can give consent on behalf of another adult.*
 - *Under these circumstances medical staffs are required to act 'in the patient's best interests'.*
 - This will mean taking into account not only the benefits of the proposed treatment, but also any views previously expressed by the patient (e.g. refusal of blood transfusion by a Jehovah's Witness).
 - This will often require discussion with the relatives, and this opportunity should be used to inform them of the proposed treatment and the rationale for it.
 - All decisions and discussions must be clearly documented in the patient's notes.
 - Where treatment decisions are complex or not clear cut, it is advisable to obtain and document independent medical advice.
- **What constitutes evidence of consent?**
 - Most patients will be asked to sign a consent form before undergoing a procedure.
 - However, there is no legal requirement for such before anaesthesia or surgery (or anything else); the form simply shows evidence of consent at the time it was signed.
 - Consent may be given verbally and this is often the case in anaesthesia.
 - It is recommended that a written record of the content of the conversation be made in the patient's case notes.
- **What do I have to tell the patient?**
 - In obtaining consent it is essential the patient is given an adequate amount of information in a form that they can understand.
 - This will vary depending on the procedure, but may include:

- **The environment of the anaesthetic room and who they will meet**, particularly if medical students or other healthcare professionals in training will be present.
- Establishing **intravenous access and IV infusion**.
- The need for, and type of, any **invasive monitoring**.
- What to **expect during the establishment of a regional technique**.
- Being conscious throughout surgery if a regional technique alone is used, and **what they may hear**.
- **Preoxygenation**
- **Induction of anaesthesia**: although most commonly intravenous, occasionally it may be by inhalation.
- Where they will **'wake up'**: This is usually the recovery unit, but after some surgery it may be the ICU or HDU. In these circumstances the patient should be given the opportunity to visit the unit a few days before and meet some of the staff.
- **Numbness and loss of movement** after regional anaesthesia.
- The possibility of **drains, catheters and drips**: their presence may be misinterpreted by the patient as indicating unexpected problems.
- The possibility of a need for **blood transfusion**.
- **Postoperative pain control**, particularly if it requires their co-operation; for example a patient-controlled analgesia device.
- Information on **any substantial risks** with serious adverse consequences associated with the anaesthetic technique planned.
- Although the anaesthetist will be the best judge of the type of anaesthetic for each individual, patients should be given an explanation of the choices, along with the associated benefits and risks in terms they can understand.
- Most patients will have an understanding of general anaesthesia — the injection of a drug, followed by loss of consciousness and lack of awareness throughout the surgical procedure.
- If regional anaesthesia is proposed, it is essential that the patient understands and accepts that remaining conscious throughout is to be expected, unless some form of sedation is to be used.
- Most patients will want to know when they can last eat and drink before surgery, if they are to take normal medications and how they will manage without a drink.
- Some will expect or request a premed and in these circumstances the approximate timing, route of administration and likely effects should be discussed.
- Finally, before leaving ask if the patient has any questions or wants anything clarified further.
- **Who should get consent?**
 - From the above it is clear that the individual seeking consent must be able to provide the necessary information for the patient and be able to answer the patient's questions.
 - This will require the individual to be trained in, and familiar with, the procedure for which consent is sought, and is best done by a senior clinician or the person who is to perform the procedure.
 - With complex problems consent may require a multidisciplinary approach.

CHAPTER 2. PREMEDICATION

- **Premedication** originally referred to drugs administered to facilitate the induction and maintenance of anaesthesia (literally, preliminary medication). Nowadays, premedication refers to the administration of any drugs in the period before induction of anaesthesia.

- **The 6 As of premedication**

- *Anxiolysis*

- *Amnesia*

- *Antiemetic*

- *Antacid*

- *Anti-autonomic*

- *Analgesia*

1. ANXIOLYSIS

- The most commonly prescribed drugs are the **benzodiazepines**.
- They produce a degree of **sedation and amnesia**, are well absorbed from the gastrointestinal tract and are usually given orally, **45–90mins preoperatively**.
- Those most commonly used include **Temazepam 20–30mg, Diazepam 10–20mg and Lorazepam 2–4mg**. In patients who suffer from excessive somatic manifestations of anxiety, for example tachycardia, **beta blockers** may be given.
- A preoperative visit and explanation is often as effective as drugs at alleviating anxiety, and sedation does not always mean lack of anxiety.

2. AMNESIA

- Some patients specifically request that they not have any recall of the events leading up to anaesthesia and surgery. This may be accomplished by the administration of **Lorazepam** (as above) to provide anterograde amnesia.

3. ANTIEMETIC

- Reduction of nausea and vomiting
- Nausea and vomiting may follow the administration of opioids, either pre- or intraoperatively. Certain types of surgery are associated with a higher incidence of postoperative nausea and vomiting (PONV), for example gynaecology.
- Unfortunately, none of the currently used drugs can be relied on to prevent or treat established PONV.

4. ANTACID

- To modify pH and volume of gastric contents. Patients are starved preoperatively to reduce the risk of regurgitation and aspiration of gastric acid at the induction of anaesthesia (see below). This may not be possible or effective in some patients:
 - Those who require emergency surgery;
 - Those who have received opiates or are in pain will show a significant delay in gastric emptying;
 - Those with a hiatus hernia, who are at an increased risk of regurgitation.
- A variety of drug are used to try and increase the pH and reduce the volume.
 - **Oral sodium citrate (0.3M)**: 30mL orally immediately preinduction, to chemically neutralize residual acid.
 - **Ranitidine (H_2 antagonist)**: 150mg orally 12 hourly and 2 hourly preoperatively.
 - **Metoclopramide**: 10mg orally preoperatively. Increases both gastric emptying and lower oesophageal sphincter tone. Often given in conjunction with ranitidine.
 - **Omeprazole (proton pump inhibitor)**: 40mg 3–4 hourly preoperatively.
- If a naso- or orogastric tube is in place, this can be used to aspirate gastric contents.

5. ANTIAUTONOMIC

- Reduce salivation (antisialogogue), for example during fibreoptic intubation, surgery or instrumentation of the oral cavity or ketamine anaesthesia.
- **Anticholinergic**
 - Reduce the vagolytic effects on the heart, for example before the use of suxamethonium (particularly in children), during surgery on the extra ocular muscles (squint correction), or during elevation of a fractured zygoma.
- **Antisymphathomimetic effects**
 - **Atropine and hyoscine** have now largely been replaced pre- operatively by **Glycopyrrolate 0.2–0.4mg IM**.
 - Many anaesthetists would consider an IV dose given at induction more effective.
 - Increased sympathetic activity can be seen at intubation, causing tachycardia and hypertension. This is undesirable in certain patients, for example those with ischaemic heart disease or raised intracranial pressure. These responses can be attenuated by the use of beta blockers given preoperatively (e.g. atenolol, 25–50mg orally) or intravenously at induction (e.g. esmolol). Peri-operative beta blockade may also decrease the incidence of adverse coronary events in high risk patients having major surgery. An alternative is to give a potent analgesic at induction of anaesthesia, for example fentanyl, alfentanil or remifentanyl.

6. ANALGESIA

- Although the oldest form of premedication, analgesic drugs are now generally reserved for patients who are in pain preoperatively.
- The most commonly used are **Morphine, Pethidine and Fentanyl**.
- Morphine was widely used for its sedative effects but is relatively poor as an anxiolytic and has largely been replaced by the benzodiazepines.
- Opiates have a range of unwanted side-effects, including **nausea, vomiting, respiratory depression and delayed gastric emptying**.

7. MISCELLANEOUS

- A variety of other drugs are commonly given prophylactically before anaesthesia and surgery; for example:
 - **Steroids**: to patients on long-term treatment or who have received them within the past 3 months;
 - **Antibiotics**: to patients with prosthetic or diseased heart valves, or undergoing joint replacement;
 - **Anticoagulants**: as prophylaxis against deep venous thrombosis;
 - **Transdermal glyceryl trinitrate (GTN)**: as patches in patients with ischaemic heart disease to reduce the risk of coronary ischaemia;
 - **Eutectic mixture of local anaesthetics (EMLA)**: a topically applied local anaesthetic cream to reduce the pain of inserting an IV cannula.
- **Commonly used anti-emetic drugs, dose and route of administration**

Type of drug	Example	Usual dose
Dopamine antagonists	Metoclopramide	10 mg orally or IV
5-hydroxytryptamine antagonists	Ondansetron	4–8 mg orally or IV
Antihistamines	Cyclizine	50 mg IM or IV
Anticholinergics	Hyoscine	1 mg transdermal patch

CHAPTER 3. GENERAL ANAESTHESIA

I. SEDATION AGENTS

1. MIDAZOLAM

- o A short acting water soluble benzodiazepine which at higher doses causes intense **sedation** (anaesthesia) and **retrograde amnesia**.
- o The initial dose is **0.02-0.1mg/Kg** in adults older than 60 and the chronically ill or debilitated
- o Onset of action: **30-60 seconds** with Peak action at **12min**.
- o Half life: **2hrs**; Risk: May cause **hypotension**.
- o Antidote: **Flumazenil** (caution!!! must be taken as it may have a shorter duration of action than the sedative agent, resulting in re-sedation.)

2. PROPOFOL

- o Propofol is now used for procedural sedation in many EDs worldwide.
- o **Has no analgesic property**
- o Its mechanism of action is unclear but is thought to act by potentiating the inhibitory neurotransmitters GABA and glycine, which enhances spinal inhibition during anaesthesia.
- o **Dosage:**
 - For induction of anaesthesia is **1 mg/kg initially then 0.5mg/kg every 1-2min**.
 - For maintenance of anaesthesia is **4-12 mg/kg/hour**.
 - Following intravenous injection propofol acts within **30 seconds** and its duration of action is **5-10 minutes**.
- o **SIDE EFFECTS OF PROPOFOL**
 - *Pain on injection (in up to 30%)*
 - *Hypotension*
 - *Transient apnoea*
 - *Hyperventilation*
 - *Coughing and hiccup*
 - *Headache*
 - *Thrombosis and phlebitis*

3. KETAMINE

- Ketamine is the only anaesthetic agent available that has **analgesic, hypnotic, and amnesic properties**. When used correctly it is a very useful and versatile drug.
- Ketamine acts by non-competitive antagonism of the NMDA receptor Ca^{2+} channel pore and also inhibits NMDA receptor activity by interaction with the phenylcyclidine binding site.
- **Dosage and Routes:**
- Ketamine can be used intravenously and intramuscularly.
 - o **10 mg/kg IM:** when used by this route it acts within **2-8 minutes** and has a duration of action of **10-20 minutes**.
 - o **1.5-2 mg/kg IV:** administered over a period of 60 seconds. When used intravenously it acts within **30 seconds** and has a duration of action of **5-10 minutes**.
- Ketamine is also effective when administered orally, rectally, and nasally.
- Baroreceptor function is well maintained and arrhythmias are uncommon.

• SIDE EFFECTS OF KETAMINE:

- Tachycardia
 - Nausea and vomiting
 - Increase BP, CVP, Cardiac Outpt
 - Nystagmus
 - Diplopia
 - Rash
- **Ketamine 1 – 2 mg/kg IV** is the ideal induction agent in asthmatic patients due to its **bronchodilatory effects**.
 - Intravenous ketamine given in a dissociative dose may be an effective temporizing measure to avoid mechanical ventilation in adult patients with severe asthma exacerbations.

4. ENTONOX

- Entonox is a **50/50 mix of oxygen and nitrous oxide**.
- Its main actions are **analgesia and depression of the central nervous system**.
- It is not known for certain how it works but it is postulated that it acts via the modulation of enkephalins and endorphins within the central nervous system.
- It takes approximately **30 seconds** to act and continues for approximately **60 seconds** after inhalation has ceased.
- Entonox is stored in **white or blue cylinders with blue and white shoulders**
- **Indications of entonox:**
 - As an adjuvant to general anaesthesia
 - As an analgesic during labour
 - As an analgesic during painful procedures
- **SIDE EFFECTS OF ENTONOX**
 - Nausea and vomiting (15% of patients)
 - Dizziness
 - Euphoria
 - Inhibition of vitamin B12 synthesis
- **Entonox should be avoided in patients with:**
 - Head injuries,
 - Chest injuries,
 - Suspected bowel obstruction,
 - Middle Ear disease,
 - Early pregnancy and
 - B12 or folate deficiency.



II. INTRAVENOUS INDUCTION AGENTS

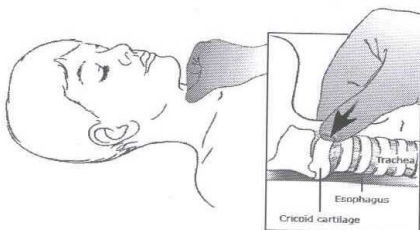
1. THIOPENTONE

- Thiopental sodium is a very short acting barbiturate that is primarily used for the induction of anaesthesia.
- Barbiturates are thought to act primarily at synapses by depressing post-synaptic sensitivity to neurotransmitters and by impairing pre-synaptic neurotransmitter release.
- The dose for **induction of anaesthesia** is **2-7 mg/kg**.
- Following intravenous injection thiopental sodium rapidly reaches the brain and causes unconsciousness within **30-45 seconds** and the effects last **5-15 minutes**.
- Its effects are cumulative with repeated administration.
- Thiopental sodium is negatively inotropic, **decreases cardiac output by approximately 20%**. It also **decreases systemic vascular resistance**.
- It is potent respiratory depressant and a period of apnoea may occur after administration.
- It also decreases renal blood flow and increases vasopressin secretion, resulting in a **fall in urine output**.
- **INDICATIONS**
 - Induction of anaesthesia
 - Treatment of status epilepticus
 - Brain protection
- **SIDE EFFECTS OF THIOPENTAL SODIUM**

○ Hypersensitivity reactions	○ Negative inotrope	○ Accumulation -> inability to assess neurology in a timely manner
○ Laryngospasm	○ Arrhythmias	○ Extravasation -> necrosis
○ Bronchospasm	○ Hypokalemia,	○ Porphyrigenic
○ Increased infection risk	Hyponatraemia	○ Cough and Headache
	○ Myocardial depression	

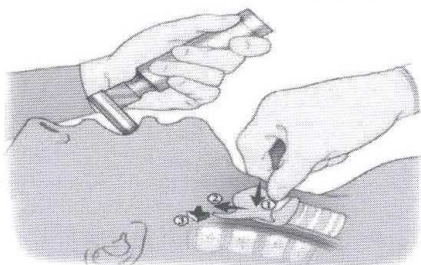
THE SELICK MANOEUVRE

- It is cricoid pressure applied during endotracheal intubation.
- It is used to reduce the risk of regurgitation of gastric contents and works by virtue of the cricoid pressure occluding the oesophagus, which passes directly behind it



BURP MANOEUVRE

- It is used to improve the view of the glottis during laryngoscopy (not to prevent regurgitation like The Sellick Manoeuvre).
- The 'BURP' manoeuvre requires an assistant to apply pressure of the thyroid cartilage posteriorly (1), then upwards (2), and finally laterally towards the patients right (3).



2. ETOMIDATE

- It is an intravenous induction agent associated with a **rapid recovery**.
- The dose for induction of anaesthesia is **0.3 mg/kg**.
- Following intravenous injection etomidate acts in **10-65 seconds** and its duration of action is **6-8 minutes**.
- Its effects are non-cumulative with repeated administration.
- Etomidate is notable for its relative **cardiovascular stability**. It causes less hypotension than thiopental sodium and propofol during induction. It is also associated with rapid recovery without a hangover effect.
- Etomidate is a potent inhibitor of steroidogenesis. Adrenal 11 beta-hydroxylase and cholesterol cleavage enzymes are inhibited by the drug, resulting in depression of cortisol and aldosterone synthesis for 24 hours after administration. *Because of this adrenocortical suppression it should not be used for maintenance of anaesthesia.*
- **SIDE EFFECTS:**
 - Adrenocortical suppression
 - Nausea and vomiting
 - Pain on injection (in up to 50%)
 - Phlebitis and venous thrombosis
 - Arrhythmias and heart block
 - Hyperventilation
 - Respiratory depression and apnoea
 - Can cause both hypo- and hypertension
 - Increased mortality in critically ill patients

3. KETAMINE AND PROPOFOL

- See previous section.

Drug	Induction and recovery	Main unwanted effects	Notes
Thiopental	Fast onset (accumulation occurs, giving slow recovery), Hangover	CVS and Resp depression	Used as induction agent declining. ↓CBF and O ₂ consumption Injection pain
Etomidate	Fast onset Fairly fast recovery	Excitatory effects during induction Adrenocortical suppression	Less CVS and resp depression than Thiopental. Injection pain
Propofol	Fast onset Fast recovery	CVS and Resp depression Pain at injection site	Most common induction agent Rapidly metabolized Possible to use as continuous infusion Injection pain Antiemetic
Ketamine	Slow onset After effects common during recovery	Psychotomimetic effects following recovery Postop Nausea-vomiting	Produces good analgesia and amnesia No injection site pain
Midazolam	Slower onset than other agents	Minimal CVS and resp depression	Little Resp and CVS depressions, No pain, good amnesia

III. INHALATIONAL INDUCTION OF ANAESTHESIA

- Breathing an inhalational anaesthetic in oxygen or in a mixture of oxygen and nitrous oxide can be used to induce anaesthesia.
- As the concentration of the anaesthetic in the brain increases slowly, unconsciousness occurs but more slowly than with an IV drug.
- Adequacy ('depth') of anaesthesia is assessed (and overdose avoided) using clinical signs or 'stages of anaesthesia'; the original description was based on using ether, but the main features can still be seen using modern drugs.
- The signs are modified by the concurrent administration of opiate or anticholinergic drugs.
 - An alternative method of inducing anaesthesia is with a volatile agent, e.g. **sevoflurane**.
 - The concentration of volatile delivered is gradually increased with the patient spontaneously breathing.
 - Common uses include paediatric practice, cases of difficult airway, difficult venous access or inhaled foreign body where maintaining spontaneous ventilation is preferable.
 - Intubation of the trachea can be achieved under deep inhalational induction without muscle relaxation.
- Currently, **sevoflurane** is the most popular anaesthetic used for this technique. Inhalation induction of anaesthesia is used when IV induction is not practical, for example in:
 - *A patient with a lack of suitable veins;*
 - *An uncooperative child;*
 - *Patients with a needle phobia;*
 - *Patients with airway compromise, in which an IV drug may cause apnoea, and ventilation and oxygenation become impossible, with catastrophic results.*



IV. MUSCLE RELAXATION

- If intubation is required, it may be necessary to paralyse the patient using:
 - **Depolarizing muscle relaxants** (e.g. suxamethonium)
 - **Non-depolarizing muscle relaxants** (Rocuronium, Cistracurium, Vecuronium or Atracurium).

1. SUXAMETHONIUM

- A short acting **depolarising muscle relaxant** with a rapid onset of action
- **Suxamethonium:**
 - Binds to the postsynaptic acetylcholine receptors, resulting in transient receptor agonism and muscle contraction followed by a refractory period of muscle relaxation within **30–60 seconds** lasting several minutes. Its relatively short-lived effects are the result of its metabolism by **Plasma Cholinesterase**.
- Dosage intravenously is **0.5–2 mg/kg**. If second dose required – **consider atropine pre-treatment**.
- Onset of action **45–60 seconds** usually preceded by fasciculation within 15 seconds.
- Initial return of muscle activity occurs within **3–5 minutes** and adequate spontaneous ventilation within **8–10 minutes**. May cause hypotension and bradycardia (after second dose, in younger children (atropine pre-treatment), in the presence of hypoxia).
- **SIDE EFFECTS OF SUXAMETHONIUM**
 - *Hyperkalemia*
 - *Malignant hyperthermia*
 - *Muscle pain*
 - *Cardiac arrhythmias*
 - *Rapid increase in intraocular pressure*
- **CONTRAINDICATIONS**
 - **Recent burns** but can be given in the first 24 hours following the burn.
 - **Spinal cord trauma** causing paraplegia. It can be given immediately after the injury but should be avoided from approximately day-10 to day-100 after the injury.
 - Other contraindications to the use of suxamethonium include:
 - *Severe muscle trauma*
 - *Hyperkalaemia*
 - *History of malignant hyperthermia*

2. ATRACURIUM

- Atracurium is a **non-depolarising neuromuscular blocker** that is used to induce muscle relaxation and paralysis to facilitate intubation and controlled ventilation.
- Atracurium competes with acetylcholine for nicotinic (N2) receptor binding sites at the post-synaptic membrane of the neuromuscular junction.
- This prevents acetylcholine from stimulating the receptors. Because the blockade is competitive muscle paralysis occurs gradually.
- In order to enhance neuromuscular recovery post nondepolarizing relaxation at the end of surgery, the amount of acetylcholine in the synapse is increased by inhibiting the **acetylcholinesterase enzyme** using a reversal agent such as **Neostigmine**.
- The 'intubating' dose of atracurium is **0.3–0.6 mg/kg** and subsequent doses are one-third of this amount.
- Satisfactory intubating conditions are produced within **90 seconds** of administration.

- There is a linear relationship between the dose and the duration of action and atracurium is non-cumulative with repeated administration.
- **The duration of action of atracurium is prolonged by the following factors:**
 - Hypocalcaemia
 - Hypokalaemia
 - Hypoproteinaemia
 - Hypercapnia
 - Hypermagnesaemia
 - Dehydration
 - Acidosis
- **Histamine release** may occur if doses $> 600 \mu\text{g/kg}$ are used. This can result in cutaneous flushing, hypotension and bronchospasm.
- **Bradycardia** has also been reported.

V. REVERSAL OF MUSCLE RELAXANTS

1. ANTICHOLINESTERASE AGENTS.

- **Neostigmine, Edrophonium, and Pyridostigmine** are used to reverse neuromuscular blockade.
- **Edrophonium** has a rapid onset, but is not as effective as neostigmine for deep blocks.
- **Pyridostigmine** has a slow onset, which makes it ill-suited to the reversal of intermediate-acting neuromuscular agents.

NEOSTIGMINE

- Remains the most commonly used anticholinesterase agent, although many principles can also apply to edrophonium and pyridostigmine.
- Reduces the intensity of neuromuscular blockade in a dose-dependent manner up to **0.04-0.05 mg/kg**, but higher doses have little if any additional benefit.
- The agent must be injected only when sufficient spontaneous recovery is observed.
- It is recommended to wait until there are four visible twitches following TOF stimulation before administering neostigmine. If no fade is visible, significant residual blockade is possible, but adequate reversal requires only **0.02-0.03 mg/kg of neostigmine**.
- If three or fewer twitches are visible, it is preferable to maintain anesthesia until there are four visible twitches and then give neostigmine at the usual **0.04-0.05 mg/kg doses**.
- *When the reversal agent is administered too early, recovery might be incomplete, and residual paralysis difficult to diagnose, as human senses cannot detect fade when the TOF ratio is 0.4 or greater.*

2. SUGAMMADEX

- Selective relaxant binding agent; forms a complex with the neuromuscular blocking agents **Rocuronium and Vecuronium**, and it reduces the amount of neuromuscular blocking agent available to bind to nicotinic cholinergic receptors in the neuromuscular junction
- As a result, sugammadex inactivates rocuronium molecules and indirectly decreases the intensity of neuromuscular blockade.
- Once bound, the kidney excretes the sugammadex-rocuronium complex.
- To a lesser extent, sugammadex also shows an affinity for **Vecuronium and Pancuronium**; however, it has no affinity for other neuromuscular blockers such as Succinylcholine, Atracurium, Cisatracurium, and Doxacurium.

- The recovery time following sugammadex administration is exceptionally fast, ie, approximately **2 minutes**.
- **DOSAGE**
 - **For Rocuronium and Vecuronium**
 - A dose of **4 mg/kg** is recommended if spontaneous recovery of the twitch response has reached 1-2 post-tetanic counts (PTC) and there are no twitch responses to train-of-four (TOF) stimulation following rocuronium- or vecuronium-induced neuromuscular blockade
 - A dose of **2 mg/kg** is recommended if spontaneous recovery has reached the reappearance of the second twitch (T2) in response to TOF stimulation following rocuronium- or vecuronium-induced neuromuscular blockade
 - **For Rocuronium only**
 - A dose of **16 mg/kg** is recommended if there is a clinical need to reverse neuromuscular blockade soon (~3 minutes) after administration of a single dose of **1.2 mg/kg** of rocuronium
 - The efficacy of the **16 mg/kg** dose following administration of vecuronium has not been studied

VI. ADJUNCTS TO ANAESTHESIA

PRE-TREATMENT PHASE

1. FENTANYL

- A potent synthetic opiate with a rapid onset of action and short half life.
- **Used to blunt sympathetic reflexes to laryngoscopy and the rise in ICP** associated with intubation.
- Dosage intravenously of **0.05-1mcg/kg**.
- May cause significant **respiratory depression, rigid chest syndrome** if given too rapidly and **hypotension**.

2. ATROPINE

- A competitive muscarinic antagonist, which causes vagal inhibition at the SA and AV nodes resulting in increased heart rate.
- **Used to counter reflex bradycardia** in children under 10 yrs or after repeat dose suxamethonium
- Dosage intravenously of **0.02mg/kg** **3 minutes before** administration of Suxamethonium.

Drug	Dose	Precautions
Morphine	0.05-0.20mg/Kg	Resp depression Histamine release with hypotension, N&V, itching, bronchospasm,
Ketofol 10mg/ml sol	IV 1:1 ratio; 1-3ml every 2 min until desired effect achieved	
Naloxone	1-2mg IV Additional 2-3min to a total 10mg	Clinical duration shorter than longer acting opioids
Flumazenil	0.02mg/kg -2mg over 15 sec Additional 0.2mg doses at 1min interval until desired state of consciousness achieved	Contraindicated in patients taking benzodiazepines for an extended amount of time Underlying seizure disorder In Patients on TCA

VII. MAINTENANCE OF ANAESTHESIA

- Can be achieved either by using one of a variety of inhalational anaesthetics in oxygen with or without nitrous oxide, or by an IV infusion of a drug, most commonly propofol.

1. INHALATIONAL ANAESTHESIA

- Inhalational anaesthetics are a group of halogenated hydrocarbons with relatively low boiling points. A 'vaporizer' is used to produce an accurate concentration in the inspired gas mixture. **Nitrous oxide** is the only other drug in this category. The inspired concentration of all of these compounds is expressed as the percentage by volume.
- There are two concepts that will help in understanding the use of inhalational anaesthetics: **Minimum Alveolar Concentration and Solubility**.
- Maintenance of anaesthesia refers to keeping a patient unconscious and can be achieved using inhaled volatile agents or continuous infusion of intravenous agents.
- Volatile agents are most commonly used, delivered via vaporisers found on the '**back bar**' of the anaesthetic machine which feed into the breathing circuit.
- The concentrations of the inhaled agents are measured and displayed. Expired end tidal concentration is equivalent to the alveolar concentration which in turn represents the concentration at the site of action (CNS). This gives the anaesthetist an idea of the amount of anaesthetic agent reaching the patient and the likely depth of anaesthesia.
- **THE MINIMAL ALVEOLAR CONCENTRATION** is the alveolar concentration of a volatile agent which when given alone prevents movement in 50% of healthy volunteers to a standard surgical stimulus (e.g. skin incision). The minimal alveolar concentration varies between different volatile agents inversely related to their potency (as their structures vary) and is also affected by other pharmacological and physiological variables.

2. PROPOFOL

- Intravenous maintenance of anaesthesia can be achieved with infusions of **Propofol** with or without an opioid delivered via a pump. Several pharmacokinetic models have been developed which map the theoretical body compartments among which a drug distributes. The desired plasma or effector site concentration can be dialled up alongside basic patient demographics (age, sex, weight) and the pump adjusts the rate of infusion to achieve the specified drug concentration. This is known as a target controlled infusion.
- Effective secure intravenous access is crucial. The choice of maintenance technique may be determined by surgical and patient factors and the experience of the anaesthetist.
- Total intravenous anaesthesia is often used in day surgery, neurosurgery or if patients get severe postoperative nausea and vomiting as it avoids emetogenic volatiles and enables rapid recovery with minimal hangover effect.

3. MIDAZOLAM

- See previous section.

4. MORPHINE

- A potent and effective opioid analgesic. Dosage intravenously is **initially 0.1–0.2 mg/kg** (usually 5mg initially). Repeat doses should be given at regular intervals during transfer at approx $\frac{1}{3}$ the initial dose.
- Onset of **action 2–3 minutes**, peak effect in **10–20 minutes**. May cause hypotension.
- Antidote: **Naloxone**

CHAPTER 4. POST-OP & RECOVERY ROOM CARE**I. THE RECOVERY AREA**

- The vast majority of patients recover from anaesthesia and surgery uneventfully, but a small and unpredictable number suffer complications.
- It is now accepted that all patients recovering from anaesthesia should be nursed in an area with appropriate facilities to deal with any of the problems that may arise, and by trained staff.
- Most patients will recover on a trolley capable of being tipped head-down.
- Patients who have undergone prolonged surgery, or where a prolonged stay is expected, may be recovered on their beds to minimize the number of transfers.
- Each patient should be cared for in a dedicated area equipped with:
 - Oxygen supply plus appropriate circuits for administration;
 - Suction;
 - ECG monitoring device;
 - Pulse oximeter;
 - Non-invasive blood pressure monitor.
- In addition the following must be available immediately:
 - **Airway equipment:** Oral and nasal airways, a range of endotracheal tubes, laryngoscopes, a bronchoscope and the instruments to perform a cricothyroidotomy and tracheostomy.
 - **Breathing and ventilation equipment:** Self-inflating bag-valve-masks, a mechanical ventilator and a chest drain set.
 - **Circulation equipment:** A defibrillator, drugs for cardiopulmonary resuscitation, a range of IV solutions, pressure infusers and devices for IV access.
 - **Drugs:** For resuscitation and anaesthesia.
 - **Monitoring equipment:** Transducers and a monitor capable of displaying two or three pressure waveforms, end-tidal carbon dioxide monitor and thermometer.
- Once anaesthesia is no longer required, maintenance agents can be switched off.
- Before emergence, adequate analgesia and antiemesis should be ensured and neuromuscular junction function restored if a muscle relaxant has been used.
- Like induction, emergence can be a time of physiological disturbance. As patients start to wake from anaesthesia or 'lighten' they may develop agitation, laryngospasm and breath-holding. Conventionally extubation is performed following oropharyngeal suction, once the patient is generating good tidal volumes and is awake, ensuring airway reflexes have returned and the patient will protect his/her own airway.
- In certain circumstances extubation may be performed 'deep', i.e. with the patient still under anaesthesia.
- Under anaesthesia, airway reflexes will remain suppressed, reducing the risk of coughing, laryngospasm and hypertension associated with extubation.
- This may be preferable in certain neurosurgical and cardiac patients in whom surges in intracranial or systemic blood pressure should be avoided.
- However, the airway will be unprotected against aspiration until the patient is awake.
- The recovery room is an intermediate place of safety between theatre and the ward where immediate surgical or anaesthetic complications can be detected and managed.
- Vital signs, pain scores and other potential problems such as postoperative nausea and vomiting are monitored.

II. PERIOPERATIVE CARE

• Warming

- As homeothermic mammals, our core temperature is designed to be around 36.5°C.
- Patients are susceptible to hypothermia under anaesthesia as a result of vasodilation causing redistribution of heat from core to periphery, convection, radiation (exposed areas), conduction (contact with cold metal objects), evaporation (endotracheal tube bypasses nasopharyngeal humidification, exposed moist mucosal surfaces) and loss of compensatory heatpreserving or heat-generating mechanisms, e.g. shivering.
- **Hypothermia** can cause coagulopathy, perioperative cardiac events, increased risk of postoperative infection and can prolong recovery and hospital stay.
- Exposure should be minimized and temperature monitored pre-, intra- and postoperatively.
- *Warm air devices and warmed fluids can be used to offset heat loss and maintain optimal body temperature.*

• Fluid balance

- The anaesthetist needs to be an expert in fluid resuscitation, using crystalloids, colloids and blood products where appropriate.
- The aim is to ensure good tissue perfusion and hence oxygenation. Rather than give a fixed fluid dose, monitors (e.g. oesophageal doppler) are often used and fluid challenges given to achieve a set endpoint, aiming to avoid hyper- or hypovolaemia.

• Positioning

- Patients are vulnerable to nerve and pressure point injury under anaesthesia and protection of these areas is the responsibility of the anaesthetist.
- Patients should ideally be in a neutral position with padding used to support at-risk areas.

• Awareness

- Awareness is the unplanned recall of events under anaesthesia and is often one of the complications patients fear most.
- It can be implicit or explicit, from a vague sense of having been awake through to specific memories of events and conversations respectively.
- Awareness is distressing and can lead to post-traumatic syndromes. Monitoring for awareness can include clinical observation (such as papillary dilatation, lacrimation, sweating) and measurement (heart rate, blood pressure, end tidal volatile concentration and depth of anaesthesia monitors).
- Most depth of anaesthesia monitors interpret patterns of neuronal electrical activity to deduce the level of conscious state.

• Other drugs

- Aside from the traditional triad of anaesthesia drugs, the cardiovascular system is often manipulated to offset the effects of anaesthesia or surgical stimulation.
- Heart rate may be increased by an anti-muscarinic (e.g. atropine or glycopyrrolate) or a mixed beta-adrenoceptor agonist (e.g. ephedrine) or reduced by betablockers.
- Blood pressure can be increased by vasoconstricting with an alpha 1 adrenoceptor agonist such as metaraminol or reduced with an alpha antagonist (e.g. phentolamine).

• Analgesia

- Pain relief is very important for patients and features in the triad of anaesthesia.
- Despite a patient being unconscious and unaware intraoperatively, stimulation (e.g. surgery) will still elicit a sympathetic response which analgesia can desirably attenuate.

- Appropriate analgesia is also essential for smooth emergence and comfort immediately after surgery.
- Analgesia is typically multi-modal with opioids titrated to extent of stimulation and predicted postoperative pain.
- **Safety**
 - Patient safety is crucially important.
 - The World Health Organization surgical safety checklist is a tool to attempt to make the perioperative journey safer and enhance team communication.
 - The entire team must ensure the correct patient is consented for the correct procedure and that any allergies or potential complications are acknowledged and shared among the team.
 - The World Health Organization surgical safety checklist has three components which are completed on arrival to the anaesthetic room, before the start of surgery or intervention and at the end of the procedure.

III. DISCHARGE OF THE PATIENT

- The anaesthetist's responsibility to the patient does not end with termination of the anaesthetic.
- Although care is handed over to the recovery staff (nurse or equivalent), the ultimate responsibility remains with the anaesthetist until discharge from the recovery area.
- If there are inadequate numbers of recovery staff to care for a newly admitted patient, the anaesthetist should adopt this role.
- **A patient who cannot maintain his/her own airway should never be left alone.**
- The length of time any patient spends in recovery will depend upon a variety of factors, including length and type of surgery, anaesthetic technique and the occurrence of any complications.
- Most units have a policy determining the minimum length of stay, which is usually around 30mins, and agreed discharge criteria.
- **MINIMUM CRITERIA FOR DISCHARGE FROM RECOVERY AREA**
 - *The patient has returned to their baseline level of consciousness.*
 - *Vital signs are within normal limits for that patient.*
 - *Respiratory status is not compromised.*
 - *Pain and discomfort have been addressed.*

IV. RECOVERY ROOM COMPLICATIONS

1. HYPOXAEMIA

- The presence of cyanosis is very insensitive and when detectable the arterial PO_2 will be $<8\text{kPa}$ (55mmHg), a saturation of 85%.
- The advent of pulse oximetry has had a major impact on the prevention of hypoxaemia and should be used routinely in all patients. If hypoxaemia is severe, persistent or when there is any doubt, **arterial blood gas** analysis should be performed.
- Hypoxaemia can be caused by a number of factors, either alone or in combination:
 - *Alveolar hypoventilation;*
 - *Ventilation and perfusion mismatch within the lungs;*
 - *Diffusion hypoxia;*
 - *Pulmonary diffusion defects;*
 - *A reduced inspired oxygen concentration.*

MANAGEMENT OF HYPOXAEMIA

- All patients should be given **oxygen** in the immediate postoperative period.
- Patients who continue to hypoventilate, have persistent V/Q mismatch, are obese, anaemic or have ischaemic heart disease, will require additional oxygen for an extended period of time.
- This is best determined either by arterial blood gas analysis or by using a pulse oximeter.

2. HYPOTENSION

- This can be due to a variety of factors, alone or in combination, that **reduce the cardiac output, the systemic vascular resistance or both:**
 - **Hypovolaemia:** *?continued bleeding*
 - **Reduced myocardial contractility:** *Ischaemic heart disease*
 - **Vasodilatation:** *regional anaesthesia, septic shock*
 - **Cardiac arrhythmias:** *Hypoxaemia; Hypovolaemia; Hypercarbia; Hypothermia; Sepsis; Pre-existing ischaemic heart disease; Electrolyte abnormalities; Hypo/hyperkalaemia, hypocalcaemia, hypomagnesaemia; Acid-base disturbances; Inotropes, antiarrhythmics, bronchodilators; Antidepressants in overdose.*

MANAGEMENT OF HYPOTENSION

- **Hypovolaemia**
 - Ensure adequate oxygenation and ventilation.
 - Intravenous fluid, either crystalloid or colloid, should be given, using a pressure infusor to speed administration. Consider cross-matching blood if not already done.
 - Stop any external haemorrhage with direct pressure.
 - Get surgical assistance if internal haemorrhage suspected.
 - Monitoring of the patient's central venous pressure (CVP) may be indicated if cardiac function is in question. In the presence of significant hypovolaemia do not waste time inserting a CVP line for venous access alone. The trend of the patient's acid-base status is a useful indicator of therapeutic success.
- **Reduced Myocardial Contractility**
 - Sit the patient upright. Give 100% oxygen.
 - Monitor the ECG, blood pressure and peripheral oxygen saturation.
 - If the diagnosis is unclear, a fluid challenge (**maximum 5mL/kg**) can be given and the response observed; an improvement in the circulatory status suggests hypovolaemia.

- Where there is no doubt about the diagnosis, fluids can be restricted initially and a diuretic (e.g. frusemide 20–40mg) given intravenously.
- Trends in the CVP can be monitored as a guide to therapy.
- Patients with ventricular failure are best cared for in a critical care area.
- If there is acute myocardial infarction, contractility may only improve with the use of **inotropes** in conjunction with vasodilators, and this is best undertaken on the intensive care unit (ICU). Unfortunately thrombolysis is contraindicated after surgery.
- **Vasodilatation**
 - Hypotension secondary to regional anaesthesia is corrected by the administration of fluids (**crystalloid**, **colloid**), the use of vasopressors (e.g. **ephedrine**), or a combination of both.
 - Oxygen should always be given.
 - The combination of hypovolaemia and vasodilatation will cause profound hypotension.
 - Patients developing septic shock require early diagnosis, invasive monitoring and circulatory support in a critical care area.
 - Antibiotic therapy should be guided by a microbiologist.
- **Cardiac arrhythmias**
 - Correction of the underlying problem will result in spontaneous resolution of most arrhythmias.
 - Specific intervention is required if there is a significant reduction in cardiac output and hypotension.

3. HYPERTENSION

- This is most common in patients with pre-existing hypertension. It may be exacerbated or caused by:
 - *Pain*
 - *Hypoxaemia*
 - *Hypercarbia*
 - *Confusion or delirium*
 - *Hypothermia*.
- A coexisting tachycardia is particularly dangerous in the presence of ischaemic heart disease as this may cause an acute myocardial infarction.
- If the blood pressure remains elevated after correcting the above, a **vasodilator** or **beta blocker** may be necessary. Senior help should be sought

4. POSTOPERATIVE NAUSEA AND VOMITING (PONV)

- This occurs in up to 80% of patients following anaesthesia and surgery. A variety of factors have been identified which increase the incidence:
 - **Age and sex:** more common in young women and children.
 - **Site of surgery:** abdominal, middle ear or the posterior cranial fossa.
 - Giving **opioid** analgesics pre-, intra- and post-operatively.
 - **Anaesthetic drugs:** etomidate, nitrous oxide.
 - **Gastric dilatation**, caused by manual ventilation with a bag and mask without a clear airway.
- Patients identified as being at risk of PONV should be given an **anti-emetic** before emergence from anaesthesia. Failure of treatment may be addressed in the recovery area by giving a second or third drug from different classes of compound.

DRUGS USED TO TREAT NAUSEA AND VOMITING

- **Antihistamines**
 - Cyclizine 50mg IM up to 6 hourly.
 - Also has anticholinergic actions; may cause a tachycardia when given IV.
- **5-HT₃ (hydroxytryptamine) antagonists**
 - Ondansetron (Zofran) 4–8mg IVI or orally, 8 hourly.
 - Has both central and peripheral actions; in the gut it blocks 5-HT₃ receptors in the mucosal vagal afferents. It does not cause dystonic movements.
- **Dopamine antagonists**
 - Metoclopramide (Maxolon) 10mg IVI/IM/PO 6 hourly.
 - Although a specific antiemetic, minimal effect against PONV. Not related to the major tranquilizers and has no sedative or antihistamine effects. Has an effect at the chemoreceptor trigger zone and increases gastric motility.
 - An alternative is Domperidone (Motilium) 10mg orally.
- **Phenothiazine derivatives**
 - Prochlorperazine (Stemetil) 12.5mg IM 6 hourly or 15–30mg orally, daily in divided doses.
 - May cause hypotension due to alpha-blockade.
 - Some have antihistamine activity and may cause dystonic muscle movements.
- **Anticholinergic drugs**
 - Atropine and hyoscine; the latter is available as a transdermal patch.
 - Severe side-effects, particularly dry mouth and blurred vision.
- **Steroids**
 - Dexamethasone 8mg IV may be useful in resistant cases.

V. POSTOPERATIVE IV FLUID THERAPY

- Oral intake should be encouraged as not all patients require routine IV fluids after anaesthesia and surgery.
- For those that do, the volume and type of fluid will be determined by a variety of factors, including:
 - *The site of surgery;*
 - *The extent of tissue damage;*
 - *Blood loss during and after surgery;*
 - *Any delay in starting to drink;*
 - *Continuing losses from the gastrointestinal tract.*
- A wide range of fluids are available and for each patient the type and volume will be dependent upon the calculated maintenance requirements of water and electrolytes plus the replacement of any abnormal losses.
- This is complemented by clinical evaluation of the patient to ensure that they are adequately hydrated, as assessed by degree of thirst, moisture of mucous membranes, blood pressure, pulse, peripheral circulation and an adequate urine output.
- In complex cases, monitoring the trend of the CVP may also prove useful

CHAPTER 5. RESPIRATORY AND CARDIAC ARREST

I. ACUTE AIRWAY OBSTRUCTION

• CONSCIOUS PATIENT

- Usually distressed and unwilling to lie down.
- Marked respiratory effort, using accessory muscles (e.g. sternomastoids).
- Indrawing of intercostal and supraclavicular regions and a tracheal tug.
- Stridor. If inspiratory, consider obstruction above the larynx.
- Tachycardia and hypertension secondary to hypoxia and hypercarbia.
- The history, if available, may indicate the cause; for example inhaled foreign body, oedema (allergic reaction, inhalational injury), infection (epiglottitis), tumour or trauma.

• UNCONSCIOUS PATIENT

- Usually secondary to unrelieved obstruction causing hypoxaemia and hypercarbia.
- Minimal respiratory effort.
- Paradoxical movement of the chest and abdomen (see saw ventilation).
- Minimal or no breath sounds (silent chest).
- Hypotension, a variety of arrhythmias, cyanosed
- **After induction of anaesthesia**
 - Obstruction apparent on attempting manually to ventilate the patient, due to a supraglottic lesion acting as a 'ball valve'.
- **During anaesthesia**
 - Mechanical obstruction of the breathing system.
 - Intrinsic airway obstruction (e.g. bronchospasm).
 - Extrinsic obstruction (e.g. tension pneumothorax).

MANAGEMENT OF ACUTE AIRWAY OBSTRUCTION IN THEATRE

- Whatever the circumstances, the aim is to secure a patent airway to allow adequate oxygenation:
 - Increase the inspired oxygen concentration to 100%.
 - Get help urgently.
 - If not already available, request the emergency airway equipment.
- **IN THE CONSCIOUS PATIENT:**
 - If safe to do so, **transfer rapidly to the anaesthetic room in theatre.**
 - If ventilation is reasonable, **induce anaesthesia** using an inhalational anaesthetic in oxygen. Increasing the inspired concentration too rapidly may cause coughing and worsen the obstruction.
 - Gentle manual supplementation of ventilation may be possible.
 - When anaesthesia is adequate **perform direct laryngoscopy.**
 - **Intubate** if possible.
 - If this fails, and the airway is adequate, carry out **formal tracheostomy.**
 - If this fails, and the airway is inadequate, carry out **needle cricothyroidotomy.**
 - Under some circumstances it may be safer initially to carry out needle cricothyroidotomy under local anaesthesia to allow oxygenation before inducing anaesthesia and direct laryngoscopy.
- **IN THE UNCONSCIOUS PATIENT:**
 - Attempt ventilation with 100% oxygen.

- Perform direct laryngoscopy quickly, **once only**.
- *If possible:*
 - Remove any foreign bodies under direct vision using Magill's forceps.
 - Pass a small-diameter (5.0mm) tracheal tube past the obstruction into the larynx.
- If this fails, proceed rapidly to either **needle or surgical cricothyroidotomy**.
- Once oxygenation is restored, the patient may recover consciousness rapidly.
- Sedation and neuromuscular blocking drugs may be required while the airway is formally assessed.
- **IN THE ANAESTHETIZED PATIENT**
 - Attempt to ventilate with 100% oxygen.
 - Perform direct laryngoscopy.
 - If possible pass a small-diameter (5.0mm) tracheal tube past the obstruction into the larynx.
 - If unsuccessful, proceed to **cricothyroidotomy**.

II. ACUTE SEVERE ASTHMA

Refer to **MAJOR PRESENTATION SECTION, CHAPTER 10 - I**

III. TENSION PNEUMOTHORAX

- A pneumothorax exists when any gas accumulates in the pleural cavity.
- This leads to collapse of the underlying lung as a result of perfusion but no ventilation ($V/Q < 1$) and hypoxaemia.
- If the gas accumulates under pressure, then a tension pneumothorax exists.
- In addition to hypoxaemia, the increasing pressure causes the mediastinum to shift, impeding venous return, severely reducing the cardiac output.
- If unrelieved, it is rapidly followed by cardiovascular collapse and death.
- **CHARACTERISTICS OF A TENSION PNEUMOTHORAX**
 - The conscious patient will be **tachypnoeic and in severe respiratory distress**.
 - In addition, and in the anaesthetized patient, signs on the affected side will include:
 - *Reduced movement;*
 - *Hyperresonance on percussion;*
 - *Decreased air entry;*
 - *Distension of the hemithorax.*
 - There may also be:
 - *Surgical emphysema;*
 - *Tachycardia, hypotension;*
 - *Deviation of the trachea away from the affected side;*
 - *Distended neck veins (if the patient is not hypovolaemic);*
 - *A gradual rise in the inflation pressure, if the patient is being ventilated, or failure to deliver a preset tidal volume.*
- **CAUSES OF TENSION PNEUMOTHORAX**
 - Puncture of the pleura lining the surface of the lung (visceral pleura). This can be due to:
 - **Chest trauma** with associated rib fractures;
 - **Insertion of central venous line**, particularly when the subclavian route is used;

- **Use of local anaesthetic nerve blocks**, for example intercostal nerves, supraclavicular brachial plexus block;
- **Rupture of an emphysematous bulla.**
- The concurrent use of positive pressure ventilation will increase the rate at which the pressure rises as gas is forced through the defect into the pleural cavity, resulting in rapid cardiovascular collapse.
- **A simple pneumothorax can tension when nitrous oxide is given:** *The nitrous oxide diffuses into the air-filled space in a greater volume and at a rate faster than nitrogen can escape, causing expansion and a rise in the pressure.*

MANAGEMENT OF TENSION PNEUMOTHORAX

- **If using nitrous oxide**, this should be **discontinued**.
- Increase the inspired oxygen concentration to **100%**.
- **Insert a 14 or 16 gauge cannula** in the second intercostal space, midclavicular line (**immediately above the third rib to avoid the neurovascular bundle**).
- If successful, this is often accompanied by an obvious release of gas under pressure, decreased respiratory distress (or inflation pressures) and an improvement in the cardiac output.
- The insertion of a cannula has the effect of converting the tension pneumothorax to a simple pneumothorax. ***This can then be treated by the insertion of a chest drain in the fifth intercostal space, midaxillary line on the affected side.***
- Finally, a **chest X-ray is taken**.
- N.B. Very rarely there may be bilateral tension pneumothoraces.

IV. SEVERE HYPOTENSION

- Hypotension is a result of a reduction in either the cardiac output or the peripheral resistance, alone or in combination (BP = COX SVR).
- Severe hypotension may be defined as a systolic pressure 40% less than the preoperative value.

A. REDUCED CARDIAC OUTPUT

- **Decreased venous return to the heart:**
 - **Hypovolaemia:** blood loss, extracellular fluid loss (diarrhoea, vomiting).
 - **Position**, for example head up, prone with abdominal compression.
 - **Mechanical obstruction impeding venous return:** pulmonary embolus, tension pneumothorax, cardiac tamponade (rare).
- **Decreased myocardial contractility:**
 - Intravenous and inhalational anaesthetic agents.
 - Ischaemic heart disease, hypoxia, hypothermia, acidosis.
 - **Extremes of heart rate:**
 - Profound bradycardia, <40 beats/min.
 - Tachycardia, >160 beats/min.

B. REDUCED PERIPHERAL RESISTANCE

- **Anaesthetic drugs:**
 - A direct action on vascular smooth muscle in the arteriolar wall, for example isoflurane.

- The release of histamine, for example atracurium.
- Reducing sympathetic tone, for example central neural block.
- **Sepsis**
 - Toxins released can cause failure of the precapillary sphincters, leading to widespread vasodilatation of the vascular bed.
- **Spinal cord injury:**
 - Injury to the cervical or upper thoracic cord causes loss of sympathetic tone to blood vessels and vasodilatation.
 - If above T5, loss of cardiac sympathetic supply will result in a bradycardia.
- **Miscellaneous:**
 - **Hypercarbia** and a **pyrexia** will both cause vasodilatation.
 - Severe hypotension requiring intervention is usually the result of a combination of the above factors; for example, vasodilatation caused by the anaesthetic drugs in a hypovolaemic patient.
- **MANAGEMENT OF HYPOTENSION**
 - Initially, time should not be spent trying to identify the cause. Treatment is symptomatic using the ABC system.
 - **Airway**
 - Clear and secure. If hypotension renders the patient unconscious, intubation will be needed to protect the airway.
 - **Breathing and ventilation**
 - Increase the inspired oxygen concentration to 100%. Support ventilation if inadequate or absent, using a **facemask initially and then via a tracheal tube**.
 - **Circulation**
 - If not already done, insert a short, wide bore cannula in the most obvious peripheral vein, often in the antecubital fossa.
 - Raise the legs to encourage venous return.
 - Fluid, either crystalloid or colloid, should be given, using a pressure infusor to speed administration.
 - Stop any external haemorrhage with direct pressure.
 - Seek urgent surgical assistance if any internal haemorrhage.
 - If a bradycardia is present (heart rate <60/min), then consider atropine 0.5–1mg IV.
- At this point, treatment should be directed towards specific causes that may be suggested by the findings on examination or by the patient's past medical history
- **ADDITIONAL MEASURES**
 - **Vasopressors:** for example **Ephedrine** to counteract vasodilatation.
 - **Inotropes:** for example **Dopamine** or **Dobutamine** to increase myocardial contractility.
 - **Antiarrhythmics.**
 - **A change in position or relief of a Tension Pneumothorax:** to allow venous return.
 - **Needle Pericardiocentesis:** in the very rare circumstances of cardiac tamponade.
 - **Monitoring of CVP or Pulmonary Artery Pressure:** to guide therapy in complex cases.

CHAPTER 6. CONTROL OF INFECTION

• PREVENTING POSTOPERATIVE INFECTION: THE ANAESTHETIST'S ROLE

- For effective prophylaxis, appropriate antibiotics should be given before skin incision as recommended by the recent WHO *Safe Surgery Saves Lives* surgical safety checklist.
- Potentially modifiable perioperative factors under control of the anaesthetist can influence the incidence of surgical site infection. Postoperative surgical patients are at risk of developing multiple types of hospital-acquired infections. These include **surgical site infections** which are relatively common (incidence 5–20%), can prolong hospital stay, cause morbidity, increase the cost of health care, and even lead to mortality.
- Other hospital-acquired infections affecting surgical patients include **respiratory and urinary tract infections**, **methicillin-resistant *Staphylococcus aureus* bacteraemias**, **antibiotic-related *Clostridium difficile* enteritis**, and **intravascular cannulae-related infections**.
- All surgical wounds are likely to become *contaminated*, usually by resident bacterial flora from skin or viscera. This may not be of clinical significance and *contaminated* wounds may go unnoticed. However, progression from wound *contamination* to *clinical infection* is largely determined by the adequacy of host defence, the most important immune mechanism of which is neutrophil phagocytosis which occurs during a crucial few hours intraoperatively and after operation.

Factors that can be optimized in the perioperative period can be divided into:

• Well-established interventions (supported by good evidence)

- Antibiotic prophylaxis,
- Hand hygiene,
- Aseptic technique during invasive procedures,
- Perioperative thermoregulation.
- Less certain interventions (some supporting evidence)
- Face masks and theatre traffic,
- Regional anaesthesia techniques,
- Inspired oxygen,
- Glycaemic control.

• Speculative interventions (no supportive evidence as yet)

- Goal-directed fluid management
- Minimizing blood transfusions,
- Enhanced recovery after surgery (ERAS),
- Avoidance of selected opioids.

1. ANTIBIOTIC PROPHYLAXIS

- Multiple randomized controlled trials and systematic reviews have consistently shown antibiotic prophylaxis to be of benefit in preventing infections after many types of surgery. The UK National Institute of Clinical Excellence (NICE) issued guidelines in 2008 recommending a single dose of prophylactic antibiotics I.V. on starting anaesthesia (i.e. before skin incision), or earlier if a tourniquet is to be used.
- **NICE recommends antibiotic prophylaxis for the following types of surgery:**
 - Clean surgery involving the placement of a prosthesis or implant,
 - Clean-contaminated surgery,
 - Contaminated surgery.

2. HAND HYGIENE

- The advent of disinfection with alcohol-based hand rub has reduced the time required to perform hand hygiene before and after every patient contact and is an accepted method to prevent transmission of resistant organisms between patients.
- The World Health Organisation is currently promoting a '5 Moments of Hand Hygiene' strategy as part of a 'Clean Care Is Safer Care' programme.
- Trust and hospital appropriate hand hygiene should be considered and practised with every single patient contact. This should be done in out-patient clinic settings, on medical ward rounds, and in the operating department.

3. ASEPTIC TECHNIQUE DURING ANAESTHETIC PROCEDURES

- Anaesthetists regularly insert central venous catheters (CVCs) and epidural catheters which may be portals of entry for bacteria.
- The incidence of CVC-related bloodstream infections is reported as high as 5–10 per 1000 catheter days.
- Guidelines in the UK, USA, and Australia recommend maximal barrier precautions for the insertion of CVCs, epidural, and nerve block catheters.
- This is often considered as part of an **'insertion bundle' approach** together with the use of **chlorhexidine antiseptics, careful selection of site, avoidance of unnecessary lines or lumens** (and prompt removal when appropriate), and **hand hygiene**.
- *They also recommend using 2% chlorhexidine in alcohol as this has higher efficacy than povidone-iodine when used for skin antiseptics.*

4. PERIOPERATIVE THERMOREGULATION

- Hypothermia triggers thermoregulatory vasoconstriction, thereby decreasing subcutaneous tissue oxygen tension. This can significantly reduce neutrophil function and collagen deposition in healing wounds.
- Hypothermia can also directly impair immune function.
- Mild perioperative hypothermia (2°C below normal core body temperature) has been shown to increase wound infection rates, delay wound healing, increase transfusion requirements, and lengthen hospital stay.
- *It is therefore recommended that patients undergoing surgical procedures for longer than 45 min have their core body temperature monitored and are actively warmed if required, except where specifically required for other surgical reasons, such as in cardiac surgery.*

5. FACE MASKS AND THEATRE TRAFFIC

- In most modern hospitals, in order to reduce rates of infections, no one is permitted to enter the operating theatre suite without wearing appropriately designated clean theatre clothing.
- Face masks, surgical scrubs, and caps form part of this theatre dress code established before the development of laminar flow operating theatres, which are known to reduce the measurable airborne bacteria surrounding the patient.
- The practice of wearing face masks is believed to minimize the transmission of oropharyngeal and nasopharyngeal bacteria from operating theatre staff to patients' wounds, thereby decreasing the likelihood of postoperative surgical site infections.

- The evidence for this was reviewed in 2001 and little was found to support or refute the routine wearing of masks by surgeons or circulating theatre personnel.
- In fact, the largest and best conducted study reviewed showed no statistically significant difference in infection rates even if the surgical team were unmasked.
- The reviewers called for further definitive studies, which have not yet been performed.

6. REGIONAL ANAESTHESIA

- Epidural analgesia results in a lower incidence of some postoperative respiratory complications, such as pneumonia, in patients undergoing laparotomy.
- This is generally considered to be as a result of superior analgesia, when compared with systemic opioids, allowing an increased ability for patients to cough and clear secretions.
- Proposed mechanisms of reduction in postoperative surgical infections are via modulation of the inflammatory response, vasodilation leading to improved tissue oxygenation, and/or improved postoperative analgesia, particularly with epidural techniques. Prospective evidence has yet to be collected in this area.

7. INSPIRED GAS COMPOSITION:

- **Oxygen vs Nitrous Oxide and Volatile Anaesthetic Agents**
 - There is some evidence that giving 80% inspired oxygen rather than 30% inspired oxygen reduces wound infections in colorectal surgery. However, this beneficial effect is not universal and other studies have failed to reproduce this effect.
 - Current guidelines to prevent surgical site infection recommend only giving the required inspired oxygen concentration to maintain oxygen saturations above 95%.

8. GLYCAEMIC CONTROL

- Acute hyperglycemia has many deleterious effects. These include:
 - Reduced vasodilation,
 - Impaired reactive endothelial nitric oxide generation,
 - Decreased complement function,
 - Increased expression of leucocyte and endothelial adhesion molecules,
 - Increased concentrations of cytokines, and impaired neutrophil chemotaxis and phagocytosis.
- These in turn could lead to increased inflammation, vulnerability to infection, and multiorgan system dysfunction.
- It has therefore been suggested that maintaining **BG below 10 mmol/dl** and reducing BG variability is likely to be both safe and effective.

9. FLUID MANAGEMENT

- Traditionally, a liberal approach to fluid infusion during surgery has been adopted in order to compensate for preoperative fasting, intraoperative fluid losses, to optimize haemodynamic variables, and maintain urine output.
- This can, however, lead to patients being significantly fluid positive perioperatively which itself can have detrimental effects.
- More recently, evidence has begun to emerge, suggesting that a more restrictive approach to fluid management reduces complications which include surgical wound site infections and other forms of sepsis (e.g. pneumonia-related).

10. ALLOGENEIC BLOOD TRANSFUSION

- **Immunomodulation and immunosuppression** are known consequences of allogeneic blood transfusion in humans.
- A meta-analysis of observational studies has also shown an association between the incidence of postoperative bacterial infection in transfused patients when compared with matched non-transfused patients.
- This effect is greater in trauma patients than surgical patients in general.

11. ERAS OR FAST-TRACK SURGERY

- This is aimed at supporting a faster recovery from surgery, earlier hospital discharge, and ultimately earlier return to normal function and quality of life.
- Many of the objectives targeted by ERAS are also likely to reduce postoperative surgical infection by reducing the duration of hospital stay, achieving early mobilization, achieving adequate analgesia, and minimizing the impact of the postoperative catabolic state.
- However, the impact on the incidence of infection rates is yet to be shown.

12. OPIOID-INDUCED IMMUNOSUPPRESSION

- The majority of opioids in current clinical practice have the propensity to suppress the immune system in humans.
- Morphine, fentanyl, remifentanyl, and meperidine, and to a lesser extent methadone have been shown to possess significant immunosuppressive properties.
- The most prevalent effects of opioid-induced immunosuppression have been reported as increased susceptibility to infection, especially after surgery, trauma, or both, decreased tolerance to malignant disease with increased likelihood of secondary deposits, and an increased chance of HIV infection in drug abusers.
- Consequently, the patient who is immunocompromised for any reason such as being on chemotherapeutic agents may be put at further risk, should an opioid with immunosuppressive activity be used as part of their treatment.

CHAPTER 7. EMERGENCY ANAESTHESIA FOR UNPREPARED PATIENTS

• SUMMARY

- Anaesthesia for emergency surgery confronts the anaesthesiologist with number of challenges.
- **Aspiration rate is the highest** in emergency cases and those with impaired consciousness.
- **Antacids and prokinetic drugs** have not been shown to improve the outcome after aspiration, and should be limited to patients at increased risk for aspiration.
- **Rapid sequence induction** is the standard of care for non-fasting emergency cases, although well-controlled trials to support this technique are lacking.
- **Trauma patients** present a particular challenge as a result of complex and life threatening injuries.
- **Hyperventilation** has been found to be associated with poor outcome.
- **Use of mannitol** has been questioned.
- **Gentle mask ventilation** should be provided in neonates, infants and sick children, in order to avoid severe hypoxemia and to allow sufficient time for skilled and atraumatic intubation in place of adult anaesthesia practice of avoiding ventilation.
- An anaesthesiologist comes across several unique challenges and providing anaesthesia for emergency surgery in unprepared patient presents one such situation.
- Limited time is available for preoperative preparation of the patient and optimization of associated medical conditions. Most commonly encountered problem is **prevention of aspiration of gastric contents** in any type of emergency surgery.

1. PREVENTION OF ASPIRATION OF GASTRIC CONTENTS

- Fasting before general anaesthesia is aimed at reducing the volume and acidity of stomach contents during surgery. Gastric emptying is delayed in emergency surgeries due **to pain, stress, elevated catecholamine levels, gastrointestinal obstruction and administration of opioids**. Hence, fasting does not ensure an empty stomach in patients coming for emergency surgeries. However, New guidelines went ahead to strictly recommend fasting status for all emergency surgeries. Likelihood of hours of fasting in these patients need to be determined by **number of hours** between intake of food and infliction of trauma/ initiation of pain. Further decision about when to operate should be based **on urgency of surgery and not hours of fasting**.
- **FACTORS REDUCING GASTRIC EMPTYING**
 - Gastric emptying may be delayed in trauma patients even if it is not abdominal trauma and is related to the **magnitude of trauma**.
 - **Decreased level of consciousness** in trauma patients: reduces tone of lower esophageal sphincter and delays gastric emptying.
 - **Hormonal changes in pregnancy** impair the tone of gastro-esophageal sphincter.
 - **Gravid uterus** impairs the position of pylorus thus further delaying the gastric emptying.
 - **Diabetic autonomic neuropathy** is associated with reduced gastric emptying.
 - **Renal failure** delays gastric emptying in dialysis patients. Gastric emptying is further delayed in patients who are both diabetic and have chronic renal failure.

- All these factors are frequently encountered in patients coming for emergency surgeries, further enhancing the risk of aspiration.
- **PREVENTIVE MEASURES AGAINST GASTRIC ASPIRATION**
 - **Preoperative starvation:**
 - A decrease in gastric acidity and facilitation of gastric drainage to reduce gastric volume have been used to decrease aspiration
 - **Decreasing gastric acidity**
 - Non-particulate oral antacid
 - H₂-receptor antagonists
 - **Reducing gastric volume**
 - Nasogastric tube
 - Prokinetic drugs
 - Cricoid pressure (**Sellick Manoeuvre**)

2. ANAESTHESIA IN UNPREPARED TRAUMA PATIENTS

- Unprepared trauma patient is seen by an anaesthesiologist for surgery to secure airway, exploratory thoracotomy, decompressive craniotomy, vascular injuries of extremities, unstable orthopaedic fractures and compound fracture.
- **Airway management**
 - In unprepared trauma patients, airway control can be achieved mainly by two ways i.e. **immediate endotracheal intubation and tracheostomy.**
 - **Indications for immediate endotracheal intubation are as follows:**
 - *Head injury with GCS<9*
 - *Shock*
 - *Airway obstruction*
 - *Combative patient requiring sedation*
 - *General anaesthesia*
 - *Chest trauma with hypoventilation*
 - **Emergency tracheostomy** is reserved for those patients whose trachea cannot be intubated transorally:
 - *Massive disruption of floor of mouth*
 - *Disruption of larynx/ cervical trachea*

A. AIRWAY CONTROL BY ENDOTRACHEAL INTUBATION

- Trauma patient who cannot maintain a patent airway/ protected should have a cuffed tube placed in trachea.
- **Rapid sequence intubation** is preferred in trauma patients.
- Sufficient help should be available to:
 - Provide inline cervical spine stabilization
 - Provide cricoid pressure
 - Mask ventilate and then intubate the patient
 - Administer drugs to the patient.
- Administering oxygen via bag and mask ventilation, using cricoid pressure to protect the airway, improves oxygenation prior to endotracheal intubation.
- **Treatment of shock**
 - Early and definitive cessation of bleeding.
 - Permissive hypotension
 - Early transfusion and Specific procoagulant therapy

- **Type of intravenous fluid:**
 - Crystalloid fluid
 - Colloid solution (no good evidence to support the use of colloid solutions)
 - Hypertonic saline
 - Hypertonic saline is gaining favor as a resuscitative fluid in trauma patients.
 - Blood products

B. ANAESTHETIC MANAGEMENT OF TRAUMA PATIENTS

• Preanaesthetic history:

- Although time constraints do not permit the detailed preoperative evaluation, whenever possible preanaesthetic history about:
 - The mechanism of injury,
 - Scene of accident,
 - Information regarding allergies,
 - Pre-existing diseases, Previous surgeries,
 - Time and type of last oral intake,
 - History of substance abuse and drug therapy should be obtained either from the patient or the attendant.
 - A history of chronic drug abuse alerts the anaesthesiologist to prepare for unusual responses to anaesthetic drugs.

• Physical examination

- It involves evaluation of:
 - Glasgow Coma Scale (GCS) for central nervous system status,
 - Upper airway for potential intubation problems,
 - Chest for signs of pneumothorax, myocardial contusion, pericardial tamponade
 - Circulatory system for signs of hemorrhagic shock.

• Intraoperative management

- As the blood volume is reduced, induction agent gets concentrated at active sites in the brain. In addition, a reduction in hepatic blood flow prolongs clearance of drugs.
- **Extremely small intravenous doses of induction agents** are so required to get desired drug effect.
- Hypnosis and amnesia are secondary to maintenance of hemodynamics and oxygenation in trauma patients.

DRUG CONTRADICTED FOR TRAUMA PATIENTS ARE:

- **Ketamine:** causes hypotension in patients with high sympathetic tone and increases intraocular pressure (IOP) which is important in patients who have associated globe injury.
- **Succinylcholine:** causes hyperkalemia if used after 24 hours of spinal cord injury or major burns. Also, it increases intracranial pressure (ICP)/IOP, practical importance of which is controversial.
- **Nitrous oxide:** is contraindicated in patients with potential closed air spaces such as pneumothorax, pneumocephaly and gas filled bowel loops.

INTRAOPERATIVE PROBLEMS ENCOUNTERED IN TRAUMA PATIENTS ARE:

- Difficult airway
- Hypothermia
- Massive blood transfusion: dilutional thrombocytopenia, prolonged clotting time.
- Hypoxia secondary to expansion of occult pneumothorax, long bone fracture and PE
- Malignant hyperthermia
- Cardiac arrest

C. TRAUMATIC BRAIN INJURY & ANAESTHESIA

- Traumatic brain injury (TBI) is common and is the leading cause of death among adults younger than 45 yr. Appropriate resuscitation and early management is the mainstay of good outcome in these patients.
- **RISK FACTORS FOR POOR OUTCOME**
 - *Severity of injury*
 - *Hypotension*
 - *Hyperglycaemia*
 - *Hypoxia*
 - *Hypercapnia and hypocapnia*
- **Mannitol**
 - There is no evidence to support the use of mannitol in head-injured patients according to the Cochrane review.
- In summary, general principles of early management include:
 - Maintenance of adequate and stable cerebral perfusion,
 - Adequate oxygenation,
 - Avoidance of hyper/hypocapnia and
 - Avoidance of hyper/hypoglycemia while avoiding iatrogenic injury.

3. ANAESTHESIA IN UNPREPARED PAEDIATRIC PATIENTS

- Paediatric patient can present for the following unprepared emergency surgeries:
 - *Foreign body with respiratory distress*
 - *Abdominal surgeries*
 - *Malrotation/ volvulus of gut or ischemic bowel*
 - *Necrotizing enterocolitis*
 - *Intestinal atresia/ bowel obstruction/bowel perforation*
- In all these situations, the major concern is full stomach.
- Classical adult type rapid sequence induction (RSI) in children is a controversial issue and it is performed with great variability.
- Further, use of suxamethonium is often debated and much effort is undertaken to find alternative approaches to achieve adequate intubation conditions **within 1 min.**
- The 1-min apnea during classical RSI is poorly tolerated in paediatric patients because of limited co-operation during preoxygenation, reduced functional residual capacity and increased oxygen demand.
- The application of gentle mask ventilation or CPAP, for prevention of hypoxia is being used by many pediatric anesthesiologists.
- However, the role of these techniques to prevent hypoxia without inducing gastric inflation and regurgitation is not discussed adequately. Even with optimal preoxygenation, apnea tolerance in the infants aged <6 months is reported to be <100 s.
- Gentle, pressure-limited mask ventilation with 100% oxygen is preferred by many anaesthesiologist to avoid hypoxia and hypercapnia.
- In children, cricoid pressure distorts airway, makes intubation difficult, relaxes lower esophageal sphincter and provokes coughing and bucking.
- Laryngoscopy under light anaesthesia elicits vomiting and aspiration.
- *So, adequate plane of anaesthesia, avoidance of cricoid pressure and complete muscle paralysis before making attempt for intubation are the key features of an appropriate paediatric rapid sequence induction.*

CHAPTER 8. AIRWAY MANAGEMENT**I. BASIC AIRWAY MANAGEMENT****1. CAUSES OF AIRWAY OBSTRUCTION (apart from smooth muscle relaxation)**

IN THE LUMEN	IN THE WALL	FROM OUTSIDE THE AIRWAY
<ul style="list-style-type: none"> • Vomit • Secretions • Blood • Foreign body 	<ul style="list-style-type: none"> • Infection, including: <ul style="list-style-type: none"> • Tonsillitis, • Peritonsillar abscess, • Retropharyngeal abscess • Floor of the mouth infection • Epiglottitis • Trauma to larynx (blunt and penetrating) • Tumour • Anaphylaxis • Angioedema 	<ul style="list-style-type: none"> • Penetrating neck injury • Tumour • Oesophageal foreign body

2. Identification of Patients with Airway Difficulty or Predicted Airway Difficulty

- **Conscious patients** with airway compromise typically sit upright intuitively:
 - o **Look** for the swollen tongue in angioedema cases, inflammation and sooty sputum following thermal injury, neck haematoma following blunt or penetrating injury, an associated rash in anaphylaxis and the increased work of breathing seen in severe asthma.
 - o **Listen** for stridor or wheeze.
 - o **Feel** for unstable facial fractures and the crepitus and surgical emphysema of laryngeal injury.
- **In unconscious patients:**
 - o **Look** for abnormal chest and abdominal wall movement, suggesting airway obstruction and the lack of fogging of the oxygen mask.
 - o **Listen** for the snoring noise of partial airway obstruction.

A. SIMPLE AIRWAY MANOEUVRES**1. SUCTION**

- o Unconscious patients are vulnerable to aspiration from vomit, blood and secretions.
- o Use gentle suction under direct vision to remove these with a wide bore rigid sucker.
- o When faced with an actively vomiting or regurgitating patient, or where there is a significant amount of blood in the airway, **turn the patient on their side and tip the trolley head down**. Turning the patient is not an option where cervical spine injury is suspected, unless you can maintain the head and neck in-line with the torso.

2. THE CHIN-LIFT MANOEUVRE

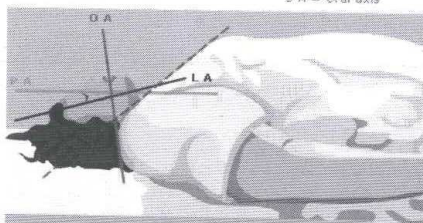
- o Unconscious patients lying supine on a trolley are vulnerable to airway obstruction.
- o Their oral axis (OA), pharyngeal axis (PA) and laryngeal axis (LA) are malaligned.
- o **Placement of a pillow or folded blanket beneath their head**, together with a chin-lift manoeuvre should improve the alignment of the axes, i.e. open up the angle between the OA and LA.
- o *The pillow effectively flexes the neck in relation to the torso; the chin-lift manoeuvre extends the head in relation to the neck. The so called 'sniffing position' is achieved.*

o The chin-lift manoeuvre obese patients:

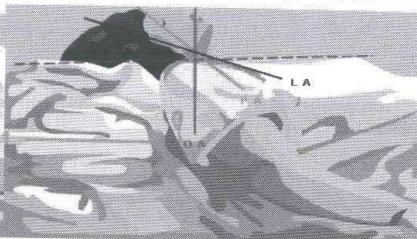
- In obese patients, especially those with short necks, the pillow may compromise the airway further by causing flexion of the head in relation to the neck.
- As a result, their chin is brought into closer proximity to their chest. This will also make subsequent intubation more difficult, if this is planned.
- The solution is **to place a pillow under the patient's shoulders and a number of pillows under their head to elevate the chin above the chest.**

L A = Laryngeal axis
P A = Pharyngeal axis
O A = Oral axis

L A = Laryngeal axis
P A = Pharyngeal axis
O A = Oral axis



Obese patient: Chin-lift manoeuvre. **Neutral position, malalignment of the axes.**



Obese patient: Chin-lift manoeuvre. **'Sniffing' position, improved alignment of the axes.**

3. THE JAW THRUST

- The jaw thrust effectively lifts the mandible forwards, lifting the tongue off the posterior pharynx at the same time.

Considerations: What manoeuvre to choose?

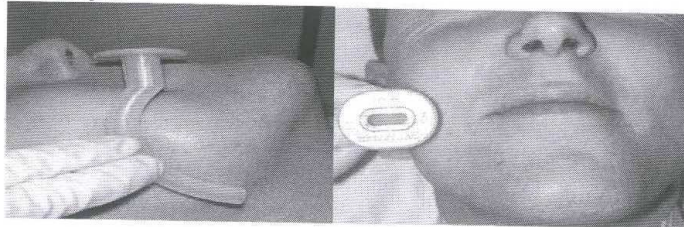
- o The **chin-lift** is suitable for those patients who, with an open airway, are breathing adequately. **A high flow oxygen mask can be applied.**
- o A **jaw thrust** is more suitable for patients who require **bag-mask ventilation**, since it is difficult to apply a mask and a chin-lift simultaneously.
- o In **trauma patients** (suspected cervical spine injury), apply the **jaw thrust** not the chin-lift manoeuvre.

B. SIMPLE AIRWAY ADJUNCTS

- The **oropharyngeal** and **nasopharyngeal** airways are designed to address airway obstruction. Both are in general tolerated only in unconscious patients. Unless you anticipate an improvement in conscious level in the short term, consider the need for intubation. **Tolerance of an oropharyngeal airway is one of the best indicators of an unprotected airway.**

1. OROPHARYNGEAL AIRWAY

- The correct size oropharyngeal airway should reach from the **patient's incisors, to the angle of the jaw.**

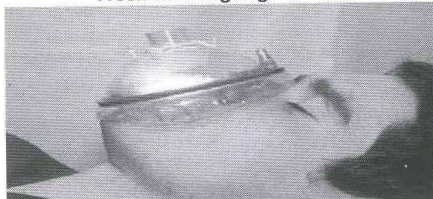


2. NASOPHARYNGEAL AIRWAY

- o The key advantage over the oropharyngeal airway is the ability of the nasopharyngeal airway to relieve airway obstruction in those patients whose mouths are difficult to open, typically patients **undergoing a seizure**. Unless it is too long, it is unlikely to stimulate the oropharynx and is better tolerated in lighter patients.
- o The disadvantage of a nasopharyngeal airway is **occasional nasal haemorrhage** as a complication, rarely profuse. Check the oropharynx post-insertion for blood.
- o *Avoid using it in patients with obvious, significant mid-face injury.*
- o The internal diameter is stamped on the side of the tube.
- o **A 6mm size for women and 7mm for men is recommended.**

C. VENTILATION

- o Having secured a patent airway, ask yourself whether the patient needs:
 - Ventilation
 - Assisted ventilation
 - An oxygen mask
- o You can subjectively gauge the adequacy of the patient's spontaneous ventilation by the depth and rate of chest wall movement.
- o If in doubt check the pCO₂ by arterial blood gas analysis.
- o If ventilation is required you will need:
 - **The correct size facemask:** one that fits snugly from the bridge of the nose to just above the chin.
 - **A self-inflating bag.**



Facemask fitting snugly from the bridge of the nose to just above the chin



Self-inflating bag

- **Adequate ventilation can be confirmed by:**
 - o *Looking for chest wall rise and fall,*
 - o *Improvement in oxygen saturation.*
- More resistance in the bag than you might anticipate suggests a problem.
- Recognise your limitations as a single-handed airway practitioner. If you sense a problem ask someone to squeeze the bag as per your instructions, whilst you attempt to provide better airway patency and mask seal using your right hand opposite your left.

DIFFICULTY IN VENTILATION

- Rarely you may find that ventilation is still difficult, in which case:
 - o Call for senior help, if you haven't already
 - o Check you have achieved optimum patient positioning
 - o Try two nasopharyngeal airways and an oropharyngeal airway
 - o If there is still no improvement, try a laryngeal mask airway
 - o If there is still no improvement allow some head and neck repositioning in trauma patients, since lack of airway patency overrides cervical spine considerations

II. DIFFICULT AIRWAY MANAGEMENT

INTRODUCTION

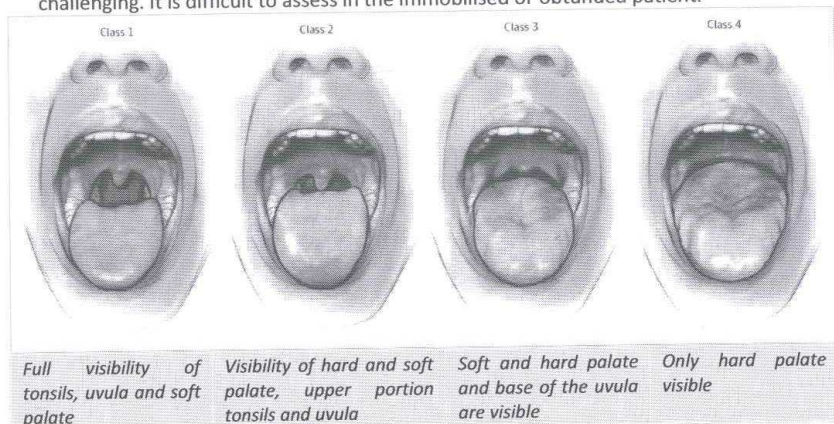
- o Airway management in an elective situation is usually straightforward. Any difficulties in airway maintenance and ventilation prior to endotracheal intubation are usually dealt with by **simple repositioning manoeuvres** and **the use of adjuncts**.
- o Laryngoscopy usually provides a clear view of the cords and intubation itself is easy.
- o In the time critical environment of the ED, the scenario is complicated by:
 - Limited scope for prior assessment
 - The often poor physiological reserve of the patient
 - The potential for a range of pathologies which may still be rapidly evolving
- o The failure rate for rapid sequence intubation in the emergency department is about 1%, with a cricothyroidotomy rate varying from 0.5% (medical patients) to 2.3% (trauma).
- **WHAT IS DIFFICULT AIRWAY?**
 - o Problems with bag-valve mask ventilation (BVM) despite repositioning and use of adjuncts, as covered in the previous session.
 - o Difficulties in intubation when attempted by a competent airway practitioner
- **FAILED INTUBATION** is the inability to successfully place an endotracheal tube after three attempts by a competent airway practitioner.
- **CAN'T INTUBATE, CAN'T VENTILATE (CICV)** is when a failed intubation is compounded by an inability to maintain adequate oxygen saturation with BVM.
- An individual's airway may be rendered difficult by:
- **Poor Preparation:**
 - o *Inadequate positioning*
 - o *Poor availability of equipment*
 - o *Lack of suitable personnel*
 - o *Inadequate training*
- The potential hazards identified, and others, have been incorporated into mnemonics from the American Emergency Airway Management course.

Difficulty Endotracheal Intubation	Difficult Bag-Mask-Valve (BMV)
<ul style="list-style-type: none"> • L Look externally • E Evaluate 3-3-2 • M Mallampati • O Obstruction/Obesity • N Neck Mobility 	<ul style="list-style-type: none"> • M Mask seal (Beard, Blood...) • O Obstructed/Obese • A Age > 55 • N No teeth/Neck Stiffness / Neck Mass • S Stridor / Snore/ Stiff Lungs
Difficult Laryngeal Mask Airway (LMA)	Difficult Cricothyrotomy
<ul style="list-style-type: none"> • R Restricted Mouth Opening • O Obstruction • D Distorted airway anatomy • S Stiff Lungs / Neck 	<ul style="list-style-type: none"> • S Surgery • H Hematoma, Have Infection (Abscess) • O Obesity • R Radiation • T Trauma, Tumor

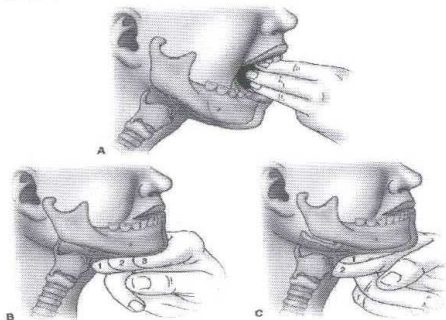
MALLAMPATI SCORE

- o Seated patient
- o Open their mouth as far as they can.
- o Using a tongue depressor or laryngoscope blade if necessary.

- o If only the base of the uvula, or less, can be visualised, intubation may be more challenging. It is difficult to assess in the immobilised or obtunded patient.



3-3-2 Rule



A normal patient should be able to accommodate:

- 3 finger breadths between incisors
- 3 fingers from the tip of the chin to the neck
- 2 fingers from the chin / neck junction to the thyroid cartilage

- The presence of indicators of possible difficulty does not mean an airway will be difficult; more importantly, their absence does not mean it will be easy.
- It is also vital to remember that pathology compromising the airway might progress rapidly. The difficulty of an airway is not a static concept.
- Pathological processes, which can compromise an airway, might develop rapidly.
- For example: **Upper airway burns**; a **mildly hoarse voice** may quickly progress to airway obstruction and the need for an emergency surgical airway. Penetrating injuries to the neck; can cause a **rapidly expanding haematoma** which may compress the airway.

DIFFICULT AIRWAY EQUIPMENTS

- A variety of new devices for airway management have been developed in recent years including:
 - o Fibreoptic stylets
 - o Video laryngoscopes
 - o Optically enhanced laryngoscopes
- They will, no doubt, have an increasingly widespread role.

MANAGEMENT OF DIFFICULT AIRWAY

- Call for help, if not already done
- Re-position patient (neck flexed 35° onto chest and 15° face extension)
- Reduce cricoid force
- Attempt 'BURP' manoeuvre
- Use gum elastic bougie
- Try alternative laryngoscopy blade: If successful >>> Tracheal Intubation
- If there is still no improvement, try a laryngeal mask airway

DISCHARGE STATUS

- Patients should be formally assessed for discharge suitability from the clinical area where sedation has taken place. Discharge criteria are as follows:
 - The patient has returned to their baseline level of consciousness.
 - Vital signs are within normal limits for that patient.
 - Respiratory status is not compromised.
 - Pain and discomfort have been addressed.
- If there is a requirement to discharge the patient prior to meeting these criteria they should be transferred to an appropriate clinical environment, usually level 2 care with continuation of periprocedure monitoring standards.
- Patients meeting discharge criteria following sedation who go on to be discharged home from the ED should be discharged into the care of a responsible third party.
- Verbal and written instructions should be given

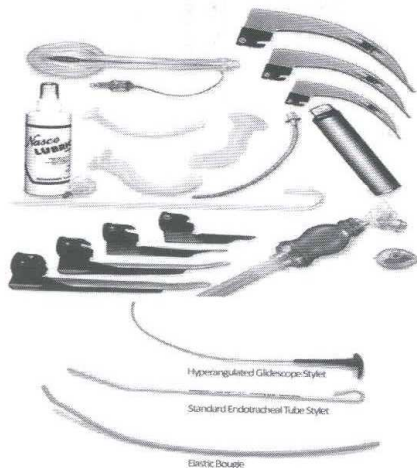
III. RAPID SEQUENCE INTUBATION (RSI)

- **MINIMUM ESSENTIAL EQUIPMENT (in addition to standard airway equipment)**

- Microlaryngoscopy endotracheal tubes, sizes 5 mm and 6 mm
- Tracheostomy tubes (cuffed), sizes 5–8 mm
- McCoy laryngoscope
- Gum elastic bougie
- Needle cricothyroidotomy set (for jet ventilation)
- Cricothyroidotomy set (for placement of tracheostomy tube)
- LMAs sizes 3–5 OR Combitube

- **DESIRABLE EQUIPMENT**

- Intubating laryngeal mask airway
- Lighted stylet
- Fibreoptic bronchoscope



- **AIRWAY/VENTILATION PROBLEMS ASSOCIATED WITH SERIOUS ILLNESS AND INJURY**

- Pre-oxygenation may be impossible or ineffective
- Positioning for intubation may be difficult if the cervical spine is immobilised
- The airway may be partially obstructed by trauma, blood, vomitus or secretions

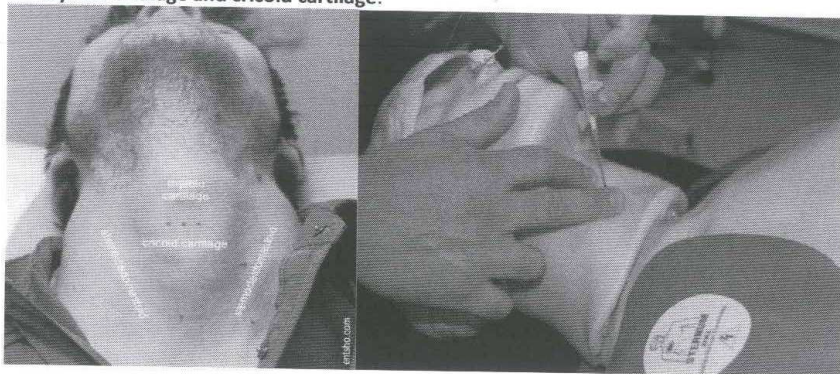
- The patient may be uncooperative
- They patient may already be hypoxic or haemodynamically compromised
- It may be impossible to predict whether the patient is likely to represent a difficult intubation
- **COMPLICATIONS OF ATTEMPTED INTUBATION IN THE EMERGENCY DEPARTMENT**
 - *Failure to intubate*
 - *Hypoxia*
 - *Unrecognised oesophageal intubation*
 - *Aspiration of stomach contents*
 - *Hypotension*
 - *Awareness*
 - *Arrhythmias*
 - *Cardiac arrest*
- **CLINICAL INDICATIONS FOR RSI**
 - **Patient who has taken an overdose:** comatose, cardiovascularly stable and maintaining a patent airway. Protection of the airway is desirable but not required immediately
 - **Isolated head injury:** Hypoxic, GCS 5; facial injury, blood in the pharynx, masseter spasm
 - **Chest injury:** requiring urgent ventilation (for example, bilateral flail segments; pulmonary contusion; drained haemopneumothoraces with hypoxia despite adequate drainage and supplemental oxygen)
 - **Asthma:** Exhausted asthmatic on maximal therapy
 - **Status epilepticus:** unresponsive to other therapy
- **CONTRAINDICATIONS OF RSI**
 - *Spontaneous breathing with adequate ventilation*
 - *Operator concern that both intubation and BVM may not be successful*
 - *Major laryngeal trauma*
 - *Distorted facial/ airway anatomy*
- **ESSENTIAL FEATURES OF RAPID SEQUENCE INDUCTION**
 - *Pre-oxygenation with 100% oxygen*
 - *Predetermined induction doses of drugs*
 - *Cricoid pressure*
 - *Cuffed endotracheal tube*
 - *Equipment and strategy to manage failed intubation*
- **DRUG DOSAGES FOR RSI**
- **IBW = ideal body weight, TBW = total body weight**
 - **INDUCTION AGENTS**
 - Ketamine 1.5-2 mg/kg IBW
 - Etomidate 0.3-0.4 mg/kg TBW
 - Fentanyl 2-10 mcg/kg TBW
 - Midazolam 0.1-0.3 mg/kg TBW
 - Propofol 1-2.5 mg/kg IBW + (0.4 x TBW) (others simply use 1.5 mg/kg x TBW as the general guide)
 - Thiopental 2-7 mg/kg TBW

- **NEUMUSCULAR BLOCKERS:**

- Suxamethonium 1-2 mg/kg TBW
- Rocuronium 0.6-1.2 mg/kg IBW
- Vecuronium 0.15-0.25 mg/kg IBW

IV. NEEDLE CRICOTHYROIDOTOMY

- Cricothyroidotomy is an important emergency procedure that can be used to obtain an airway when other, more routine methods have failed.
- Occurs at the **cricothyroid membrane** which can be palpated anteriorly between the **thyroid cartilage** and **cricoid cartilage**.



- **THE PROCEDURE IS CARRIED OUT AS FOLLOWS:**

- Assemble and prepare oxygen tubing by cutting a hole towards one end of the tubing
- Connect tubing to an oxygen source capable of delivering > 50 psi
- Surgically prepare the neck using **antiseptic swabs**
- **Palpate the cricothyroid membrane** anteriorly between the thyroid cartilage and cricoid cartilage
- **Puncture the skin** in the midline and insert 12G or 14G cannula through cricothyroid membrane at 45 degree angle caudally
- **Confirm tracheal position** by air aspiration (20ml syringe)
- **Attach ventilation system** to cannula
- Commence cautious ventilation (**1 second on and 4 seconds off**)
- **Confirm ventilation of lungs**, and exhalation through upper airway
- Ventilation can now be maintained for **30 – 45 minutes**

- **COMPLICATIONS OF NEEDLE CRICOTHYROIDECTOMY**

- *Inadequate ventilation, hypoxia and Death*
- *Aspiration (blood)*
- *Oesophageal laceration*
- *Haematoma*
- *Perforation of posterior tracheal wall*
- *Subcutaneous or mediastinal emphysema*
- *Thyroid perforation*

CHAPTER 9. PROCEDURAL SEDATION

- **INDICATIONS FOR PROCEDURAL SEDATION**

- o Procedural sedation is indicated in the ED when behavioural management and analgesia are not adequate to facilitate a procedure or examination.
- o Typically, this will be for reduction of a fracture or dislocation

- **CONTRA-INDICATIONS FOR PROCEDURAL SEDATION**

- o **Allergy or hypersensitivity** to the relevant medications
- o **Lack of appropriately trained personnel** to perform the sedation
- o **Patients have an ASA IV and above**
- o **Lack of appropriate monitoring and resuscitation equipment** (for Potential G.A.)
- o **High risk of aspiration**, e.g. acute **alcohol intoxication**

- 1. **CLINICAL ASSESSMENT**

- **AMPLE system:** This assessment should include a past medical history, drug history and focussed clinical examination to identify any existing medical illnesses, particularly cardiovascular or respiratory disease and allergy.
- It is essential that a clinical assessment is made to identify patients who:
 - o Have a ASA classification of IV or above
 - o May be difficult to ventilate

- 2. **AIRWAY ASSESSMENT**

- Using the **LEMON** method: highlights patients who may be **difficult to intubate**:
 - o **L:** Look externally (facial trauma, beard, large incisors, large tongue)
 - o **E:** Evaluate the 3-3-2 rule
 - o **M:** Mallampati score
 - o **O:** Obesity/obstruction (stridor in particular is worrying)
 - o **N:** Neck mobility
- **AIRWAY MANAGEMENT**
 - o The presence of any anatomic features that may affect airway management should be noted carefully.

- 3. **FASTING**

- Aspiration is a rare complication of procedural sedation.
- There is a paucity of evidence to make an absolute recommendation regarding minimum fasting times prior to procedural sedation; however, the following principles should be borne in mind:
 - o Protective airway reflexes are more likely to be impaired with deep sedation, making aspiration more likely in the event of regurgitation.
 - o In circumstances where life or limb are not threatened, a procedure may be delayed to ensure safer sedation without altering the clinical outcome.
 - o There is a paucity of evidence to suggest a minimum pre-sedation fasting time, however the practitioner should consider the urgency of any procedure when managing an unfasted patient.

4. PHARMACEUTICAL AGENTS

HOW DO I DECIDE WHICH PHARMACEUTICAL AGENT IS MOST APPROPRIATE?

- The ideal sedative agent should produce sedation **rapidly and reproducibly**.
- The level of sedation should be dose related, predictable and the **recovery time should be rapid**. The agent should have a **minimal effect on the cardiovascular and respiratory systems** and the therapeutic window should provide acceptable **margins of safety**.
- This ideal sedative agent does not yet exist.
- Refer to some specific sedative agents discussed earlier in this section:
 - Midazolam / Propofol / Ketamine / Nitrous oxide

DISSOCIATIVE SEDATION

- A separate sedation category, '**dissociative sedation**', has therefore been introduced.
- **Dissociative sedation** is defined as '*a trance like cataleptic state characterized by profound analgesia and amnesia, with retention of protective airway reflexes, spontaneous respirations, and cardiopulmonary stability.*'
- **Ketamine** is a unique drug in sedation practice because it causes a dissociative state that does not fit the standard definitions of sedation listed above.
- We recognise that an important boundary exists between moderate or 'conscious' sedation, where the patient responds purposefully to verbal commands, and deeper levels of sedation where the patient responds only to painful stimuli, or not at all.
- Once verbal contact with the patient is lost it becomes difficult to determine the level of unconsciousness, and over-sedation with an associated risk of airway and cardio-respiratory complications is possible.
- Deeper levels of sedation are, to all intents and purposes, indistinguishable from general anaesthesia and should therefore be treated as such. Because sedation is a continuum, it is not always possible to predict how the individual patient will respond. Patients in whom conscious sedation is intended have the potential to become more deeply sedated.
- Practitioners intending to produce a given level of sedation must therefore be able to 'rescue' patients from a deeper level of sedation than intended.
- *A clinician intending to achieve 'deep sedation' should therefore have the knowledge and skills to manage and rescue a patient from general anaesthesia.*

RECOMMENDATIONS FOR SAFE SEDATION IN THE ED

- Immediate Life Support comprises the essential knowledge and skills to enable recognition of the acutely ill patient and treatment of a patient in cardiac arrest while awaiting the arrival of a resuscitation team.
- Competencies within the domain of ILS include: delivery of high-quality chest compressions, basic airway management, safe defibrillation using either manual or automated external defibrillators (AEDs), and being a cardiac arrest team member.
- **OXYGEN**
 - Oxygen should be given to sedated patients, who may experience a fall in oxygen saturation from the baseline level measured on room air.
 - *Oxygen should be given from the start of sedative administration until the patient is ready for discharge from the recovery area.*
- **CAPNOGRAPHY**
 - The use of continuous capnography is mandatory wherever deep sedation, dissociative sedation, general anaesthesia or RSI occurs, except in rare cases where it would substantially interfere with surgical access.

- Capnography is also recommended at lighter levels of sedation; this is an emerging area of practice, and the use of capnography is expected to become routine.

• **DOCUMENTATION**

- Standard forms should be routinely used for patient pre-assessment, patient information, consent, monitoring, discharge information and clinical audit.
- Past medical history, medications, allergies and physical examination of vital signs, airway and cardiopulmonary status should all be recorded prior to the procedure.
- Good practice guidelines, issued by the Department of Health, include standard consent forms for patients undergoing procedures including sedation and general anaesthesia, but national agreement has not been established in the other documentation areas, and the development of appropriate forms would be welcomed.

• **POST-PROCEDURE MONITORING**

- *All patients who have received sedation should continue to be managed in a clinical area that provides the same level of facilities and monitoring as those required during the procedure, until the level of consciousness and other vital signs have returned to pre-procedure baseline levels.* This includes the presence of a clinician who has been trained in the core skills required of recovery nurses, as described in guidelines issued by the Association of Anaesthetists of Great Britain and Ireland.
- *These skills include **the monitoring and measurement of vital signs and overall patient status, including respiratory rate, blood pressure, heart rate, Glasgow Coma Score and basic life support training.***

• **DISCHARGE STATUS**

- Patients should be formally assessed for discharge suitability from the clinical area where sedation has taken place.
- Discharge criteria are as follows:
 - *The patient has returned to their baseline level of consciousness.*
 - *Vital signs are within normal limits for that patient.*
 - *Respiratory status is not compromised.*
 - *Pain and discomfort have been addressed.*

• **ACTING ON INCREASED ASPIRATION RISK**

- Where the risk of aspiration is significantly increased steps should be taken to mitigate this risk. Suggested approaches include:
 - **Delaying the procedure**, if clinically appropriate.
 - **Adopting an alternative technique:** Rapid sequence induction of anaesthesia and tracheal intubation is considered the 'gold standard' where there is an increased aspiration risk, but pulmonary aspiration may still occur. In addition, RSI introduces other risks, such as inability to intubate or ventilate the patient and the risk of adverse reaction to induction and neuromuscular blocking drugs.
 - **Regional anaesthetic techniques** may allow the required procedure to be performed with no or minimal sedation.
 - **Reducing the depth and duration of sedation:** This increases the risk of procedural failure, but may be appropriate in some instances.
 - **Promote gastric emptying:** administration of **Ranitidine or PPIs, Metoclopramide and Sodium Citrate** is appropriate to neutralise gastric acid and promote gastric emptying.
 - In all cases of increased aspiration risk the advice of an expert sedationist should be sought. However, there is no consensus on this subject, even among experts.

CHAPTER 10. LOCAL & REGIONAL ANAESTHESIA

I. LOCAL ANAESTHESIA

BUPIVICAINE (Marcaine) 0.25% (2.5mg/ml); 0.5% (5mg/ml)

- Longer duration of action: 3-8 hrs
- Most associated with cardiac toxicity
- Slower onset of action than adrenaline
- Max. dose: 2mg/Kg (plain and adrenaline)

PRILOCAINE 0.5%; 1%

- Used in Bier's Blocks
- Max.dose: 6mg/kg plain (Not used with adrenaline)
- Half-life: 1 hour
- Prilocaine can cause Methaemoglobinaemia as one of its metabolites O-toluidine is a strong oxidizing agent which converts the Fe²⁺ (ferrous iron) of normal haemoglobin to Fe³⁺ (ferric iron) of methaemoglobin.
- Prilocaine is also used in topical anaesthetics such as EMLA (Eutectic Mixture of Local Anaesthetics - eutectic means that mixture has lower melting temperature than its individual constituents.)
- EMLA cream contains 2.5% prilocaine and 2.5% lidocaine.

LIGNOCAINE 0.5%; 1%, 2%

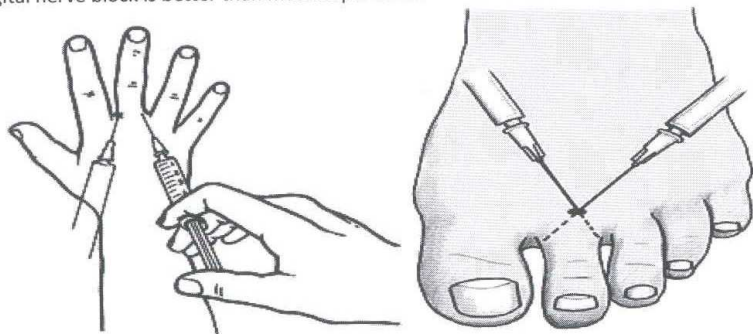
- Plain or with adrenaline 1:200,000
- Duration of action:
 - Plain: 30-60 minutes
 - With Adrenaline: 90 minutes
- Max. dose:
 - Plain: 3mg/Kg
 - With Adrenaline: 7mg/Kg
- Antidote:
 - IV infusion of a 20% Lipid Emulsion (eg, Intralipid 20%) has become an accepted part of treatment for systemic toxicity from local anesthetics
- Lidocaine 0.5% (5mg/ml)>>>mg= mls X 5)
- Each 1 ml contains 5 mg of lidocaine hydrochloride
- Lidocaine 1% (10mg/ml)>>>mg= mls X 10)
- Each 1 ml contains 10.0 mg of lidocaine hydrochloride,
- Each 20 ml solution contains 200 mg Lidocaine Hydrochloride
- Lidocaine 2% (20mg/ml)>>>mg= mls X 20)
- Each 1 ml contains 20.0 mg of lidocaine hydrochloride,
- Each 2 ml solution contains 40 mg Lidocaine Hydrochloride E.P.
- Each 5 ml solution contains 100 mg Lidocaine Hydrochloride E.P.

II. NERVE BLOCK

- o Wait longer than direct infiltration for the block to work as the anaesthetic must diffuse into larger nerves.
- o If the patient experiences pain in the nerve distribution then stop injecting immediately, withdraw slightly and try injecting again.
- o **Avoid** use Adrenaline around end arteries e.g. digital, nose, ear or penile blocks.

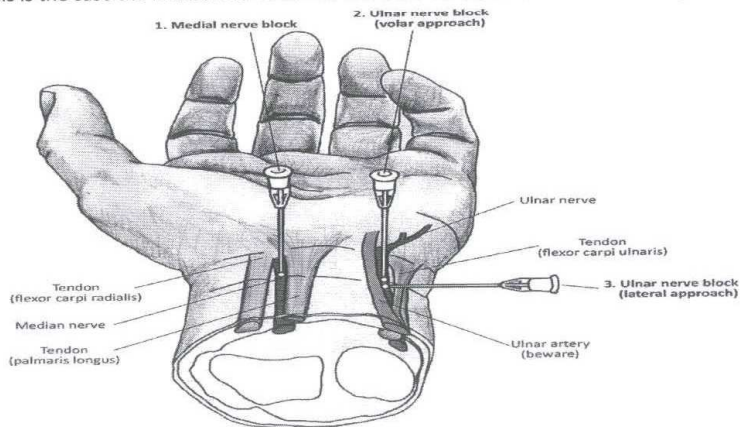
1. DIGITAL NERVE BLOCK.

- o **3mls of 0.5 % Marcaine** in preference to **1% plain lignocaine**.
- o Use either a single injection (3ml) at the **base of the finger** (MCPJ palmer crease) or two injections with a dorsal approach.
- o Usually takes 5 minutes before adequate analgesia is achieved
- o Digital nerve block is better than metacarpal block.



2. MEDIAN NERVE BLOCK

- The median nerve can be located between the tendons of **palmaris longus** and **flexor carpi radialis (PL & FCR)**.
- It should be noted that palmaris longus is absent in around 15% of the population and if this is the case the median nerve can be located **5-10 mm medial to flexor carpi radialis**.



- Once in the hand it gives off its two main sensory branches:
 - **Palmar cutaneous branch** – supplies the lateral aspect of the palm (arises in forearm and does not pass through carpal tunnel)
 - **Palmar digital branch** – supplies the palmar surface and fingertips of lateral 3 ½ digits (arises in hand)
- **LANDMARK AND PROCEDURE**
 - The needle should be inserted approximately **2.5 cm proximal to flexor retinaculum**, which can be located underneath the wrist crease.
 - In order to perform the block correctly the deep fascia, which lies 3-5 mm beneath the skin, should be penetrated.
 - Operators describe a **fascial 'click'** as the being felt as the needle passes through the fascia. The fascia is relatively thin and not always felt so many text books advice simply penetrating to a depth of 3-5 mms to ensure the fascia has been traversed and that the local anaesthetic can bathe the median nerve.
 - For the block to be effective between **5 and 10 mls of 1% lidocaine** or an equivalent does of alternative local anaesthetic should be injected.

3. ULNAR NERVE BLOCK

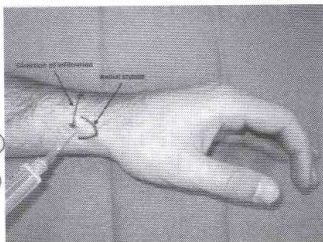
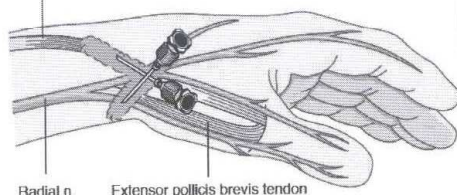
- The ulnar nerve can be located between the **ulnar artery and the tendon of flexor carpi ulnaris (FCU)**.
- The ulnar nerve lies lateral to the tendon of flexor carpi ulnaris at the wrist.
- It then passes superficial to the flexor retinaculum and enters the hand through the **ulnar canal (Guyon's canal)**.
- The sensory function of the ulnar nerve is provided by its three main sensory branches:
 - **Palmar cutaneous branch** – supplies the medial half of the palm (arises in forearm and travels into the hand)
 - **Dorsal cutaneous branch** – supplies the dorsal aspect of the medial 1 ½ digits and associated dorsal hand area (arises in forearm and travels into the hand)
 - **Superficial terminal branch** – supplies the palmar aspect of the medial 1 ½ fingers (arises in the hand)
- **LANDMARK AND PROCEDURE**
- Ulnar nerve block at the wrist can be performed as follows:
 - The needle can be inserted **medial to the ulnar artery, lateral to the tendon of flexor carpi ulnaris at the level of the wrist crease, directed towards the styloid process of the ulna**
 - Dose: between **3 and 5 mls of 1% lidocaine** or an equivalent
 - A further **2-3 mls of 1% lidocaine** can be infiltrated around the **ulnar aspect of the wrist to block the dorsal cutaneous branch**.

4. RADIAL NERVE BLOCK

- The radial nerve terminates in the forearm by dividing into two branches:
 - *The deep branch of the radial nerve and;*
 - *The superficial branch of the radial nerve*
- **The superficial branch** of the radial nerve is the primarily sensory branch in the hand.
- It descends into the forearm under **brachioradialis** running with radial artery on its medial aspect from 1/3 of the way down the forearm.
- It passes posteriorly, emerging from under the tendon of brachioradialis proximal to the radial styloid and passes over the tendons of the anatomical snuffbox.

- It then terminates as cutaneous branches to the dorsum of the hand.
- The sensory function of the radial nerve is provided by its four main sensory branches:
 - **Inferior lateral cutaneous nerve of the arm** – supplies the lateral aspect of the anterior upper arm between the deltoid and the elbow
 - **Posterior cutaneous nerve of the arm** – supplies part of the posterior aspect of the upper arm
 - **The posterior cutaneous nerve of the forearm** – supplies a tapered strip of the middle portion of the posterior forearm
 - **The superficial branch of the radial nerve** – supplies the posterior surface of the lateral 3 ½ digits and the associated areas of the palm.

Extensor pollicis longus tendon

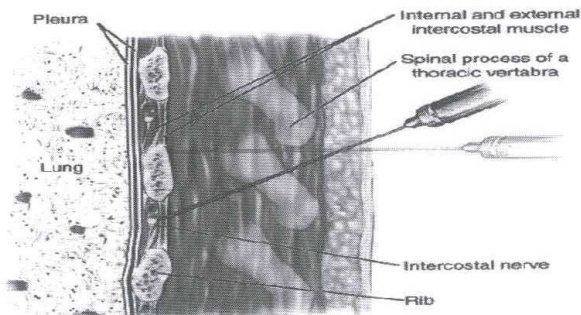


• LANDMARK AND PROCEDURE

- The block is best performed with the **wrist held in slight dorsiflexion**
- Local anaesthetic should be infiltrated subcutaneously around the **radial side and dorsum of the wrist** approximately **3 cm proximal to the radial styloid**, aiming medially towards the radial artery but with care taken not to penetrate the vessel itself.
- The infiltration can then be extended laterally. Because of the less predictable nature of local anatomy associated with this block it is essentially a '**field block**' and requires more extensive infiltration than the other nerve blocks around the wrist.

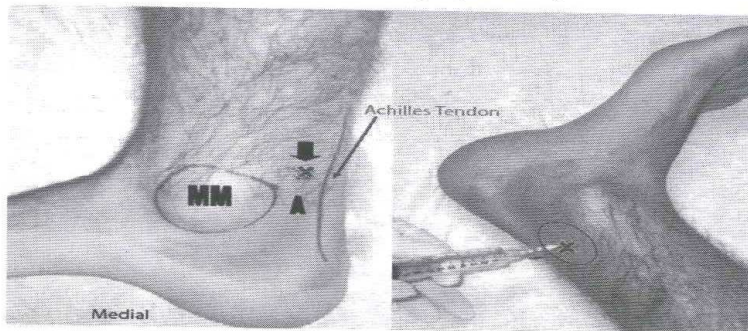
5. INTERCOSTAL BLOCK

- **Indications:** thoracic or upper abdominal surgery, rib fractures, breast surgery
- **Landmarks:** angle of the rib (6-8 cm lateral to the spinous process)
- **Needle insertion:** Under the rib with approximately 20-30° cephalad angulation
- **Target:** needle insertion 0.5 cm past the inferior border of the rib
- **Local anesthetic:** 3-5 mL per intercostal level



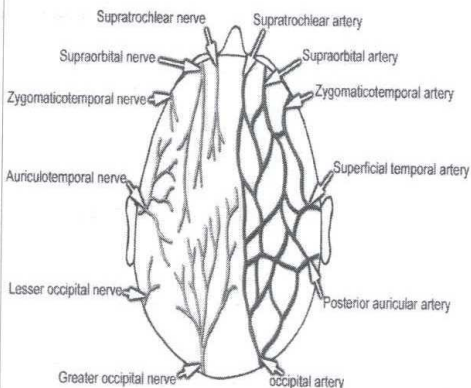
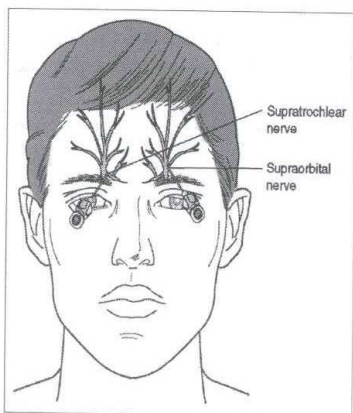
6. TIBIAL NERVE BLOCK

- For the sole of the foot.
- Use 10 ml of 1% lignocaine +/- Adrenaline.
- Do not use Adrenaline in patients with peripheral vascular disease.
- Aim **medial to the Achilles tendon behind the posterior tibia artery**, between the tendons of flexor digitorum longus and flexor hallucis longus (FDL & FHL).



7. SUPRA-ORBITAL/SUPRA-TROCHLEAR BLOCK.

- The **supraorbital nerve block** is used for forehead and scalp anaesthesia. The supra orbital nerve leaves the orbit through the supra orbital notch. This notch lies medially in the supra orbital rim, on a vertical line through the medial aspect of the cornea.
- The **supra-trochlear nerve** exits the orbit at the junction of the superior orbital rim and the medial wall of the orbit.
- The supratrochlear nerve block is often performed in conjunction with the supraorbital nerve block to achieve regional anaesthesia over the **ipsilateral forehead**. The two nerves have a sensory distribution from forehead to coronal suture from mid-line to temporal region.



- **INDICATIONS:**

- o Repair of scalp lacerations.
- o Provide anesthesia for scalp excisions, frontal craniotomies, or frontal VP shunts.
- o Can be used in conjunction with steroid injections for post-herpetic neuralgia or trigeminal neuropathies.
- **Infiltrate subcutaneously completely parallel to the supra-orbital rim above the eyebrow 5 ml of 2% lignocaine with Adrenaline.**
- Do not infiltrate directly around the nerve or its foramen.

8. INFRAORBITAL NERVE BLOCK

- **INDICATIONS**

- o Wound closure
- o Pain relief
- o Anesthesia for debridement
- o Contraindication to general anesthesia

- **CONTRAINDICATIONS**

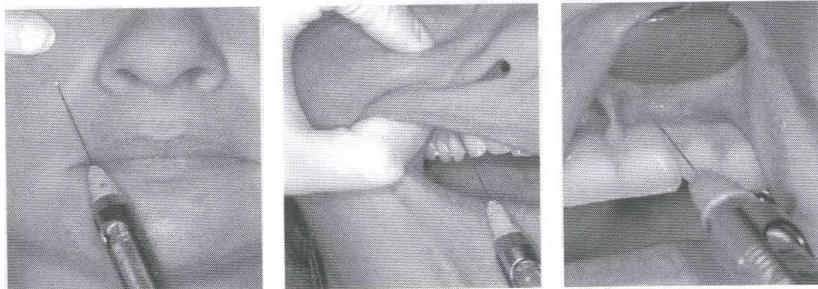
- o Any allergy or sensitivity to the anesthetic agent
- o Evidence of infection at the injection site
- o Distortion of anatomical landmarks
- o Uncooperative patient

- An infraorbital nerve block requires **1-3 mL of the chosen anesthetic agent** (Lidocaine or Bupivacaine (Marcaine) are frequently used anesthetic agent).
- The onset of action of bupivacaine is slower than that of lidocaine.

LANDMARK AND PROCEDURE

- **INTRAORAL APPROACH**

- o Apply cotton-tipped applicator soaked with topical anesthetic to the mucosa opposite the upper second bicuspid (premolar tooth) for 1 minute.
- o To palpate the infraorbital foramen, have the patient look straight ahead and *imagine a line drawn vertically (sagittally) from the pupil down toward the inferior border of the infraorbital ridge*. Keep the palpating finger in place over the inferior border on the infraorbital rim for the remaining steps.
- o Retract the cheek and introduce the needle into the mucosa opposite the upper second bicuspid approximately **0.5 cm from the buccal surface** (see images below).
- o Keep the needle parallel with the long axis of the second bicuspid until it is palpated near the foramen. (The approximate depth is 1.5-2.5 cm.)



Infraorbital nerve block, intraoral approach

- If the needle is extended too far superiorly and posteriorly, the orbit may be entered.
- Once the needle is positioned properly, aspirate to ensure that the needle is not within a vessel. Inject **2-3 mL of anesthetic solution** adjacent to the foramen.
- Take care not to inject into the foramen (which may result in swelling of the lower eyelid) by keeping the palpating finger firmly on the inferior orbital rim.

• EXTRAORAL APPROACH

- During the extraoral technique, the needle is in very close proximity to the facial artery.
- Because of this proximity, avoid adding vasoconstrictors to the anesthetic agent.
- Use the previously described landmarks to locate the infraorbital foramen.
- *Using sterile technique, insert the needle through the skin, the subcutaneous tissue, and the quadratus labii superioris muscle (see image below).*
- Aspirate to ensure the needle is not within a vessel.
- The facial artery and vein are very close to the needle in this position.
- Inject the anesthetic solution. The infiltrated tissue appears swollen.
- Firmly massage this area for 10-15 seconds.

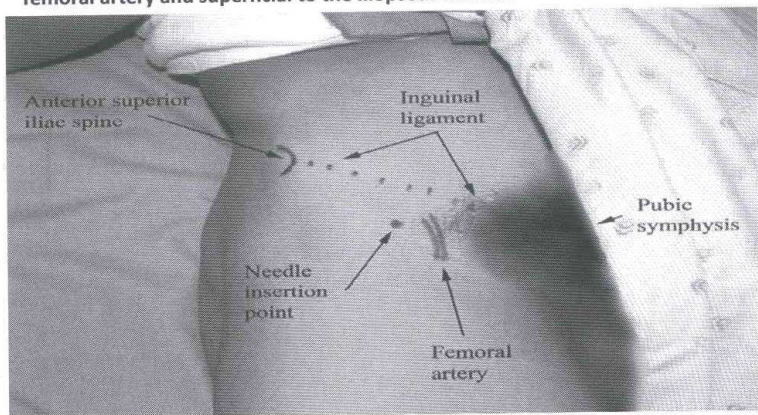


Technique for infraorbital nerve block.

9. FEMORAL NERVE BLOCK/ FASCIA ILIACA BLOCK

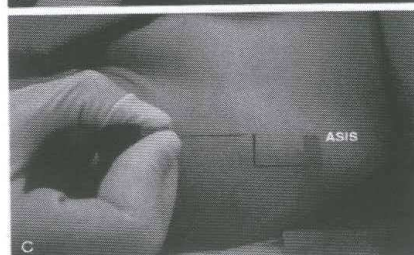
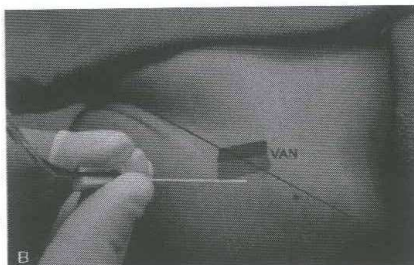
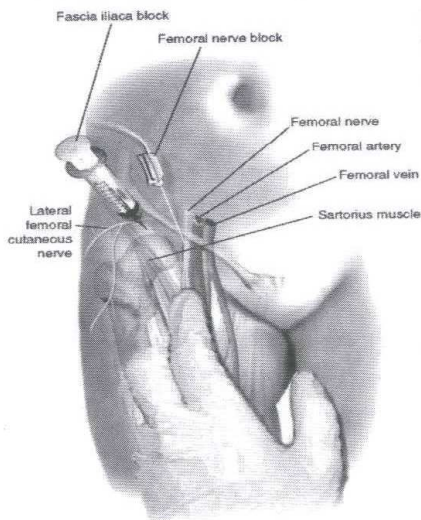
- Historically, the technique was sometimes termed the “**3 in 1 block**” because it was thought a single injection could block the **femoral, lateral femoral cutaneous and obturator nerves**. Femoral (3 in 1) block should be performed under **ultrasound guidance**
- Used for **fractured shaft of femur** or **fractures of the patella**
- If possible, obtain IV access before performing this technique
- There is a low but definite risk of local anaesthetic toxicity
- **Max dose of local anaesthetic Bupivacaine 2 mg/kg (= 0.4 ml/kg of 0.5% bupivacaine)**
- You can use a mixture of lignocaine and Bupivacaine. If mixture used, then maximum dose should be **2mg/kg in TOTAL**.
- **LANDMARKS AND SURROUNDING STRUCTURES**
 - *Important landmarks include the femoral crease, ASIS, pubic tubercle, femoral artery (palpable) and veins (not palpable), both located medially.*
- **TRADITIONAL FEMORAL BLOCK**
 - **A line is drawn from the ASIS to the pubic tubercle**, in order to outline the inguinal ligament. The femoral artery is marked.

- A 4 cm 22 ga. needle is inserted just **lateral to the femoral artery**.
- The femoral nerve is often found within a triangular hyperechoic region, **lateral to the femoral artery and superficial to the iliopsoas muscle**.



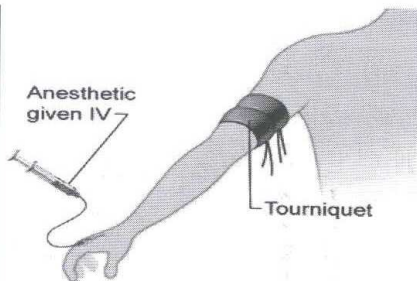
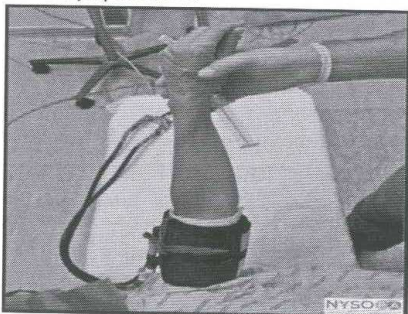
FASCIA ILIACA COMPARTMENT BLOCK

- Conduct as for Femoral Nerve block, with the following changes.
- Can be safely performed asleep as long as the target is truly lateral to the femoral nerve.
- **Use 30-40ml of local anaesthesia**, calculate the concentration based on body weight.
- Remember this block is for cutaneous analgesia, not surgical anaesthesia.
- Target for injection should be **1-2cm lateral to the lateral border of the femoral nerve**.
- Spread should be visible under the fascia iliaca, and medially to the femoral nerve.
- Much of the spread will be cephalad, and therefore not directly visible.



10. BIER'S BLOCK

- According to the 2014 RCEM best practice guideline on intravenous regional anaesthesia (IRVA or Bier's block):
 - **0.5% or 1% prilocaine** should be used (without preservative). Maximum dose of prilocaine is **6 mg/kg**
 - **0.5% lidocaine at 3 mg/kg** with a maximum dose of 200 mg (40 ml) may be used as an alternative but prilocaine remains the first line drug of choice.
 - Bupivacaine should NOT be used
- **CONTRAINDICATIONS TO PERFORMING A BIER'S BLOCK**
 - Allergy to local anaesthetic
 - Uncooperative or confused patient
 - Morbid obesity (cuff unreliable of obese arms)
 - Peripheral vascular disease, Raynaud's phenomenon
 - Severe hypertension, Scleroderma, Epilepsy
 - Sick cell disease or trait, Methaemoglobinaemia
 - Procedures needed in both arms
 - Infection in the affected limb
 - Lymphoedema



• PROCEDURE:

- Ensure patient is on a **cardiac monitor**,
- Ensure that **two doctors** are present throughout the procedure (one of which should have adequate airway management training).
- **Elevate the injured arm** for three minutes to exsanguinate the limb
- Apply and inflate the **double-cuff tourniquet** and inflate to 100mmHg above the systolic BP or to 300mmHg (whichever is greater)
- **Check for the absence of radial pulse**, Inject the **0.5%/1% plain prilocaine**
- Warn the patient about the cold/hot sensation and mottled appearance of the arm
- Check for anaesthesia, may have touch but not pain, after five minutes
- If anaesthesia inadequate, flush cannulae with 10-15 ml normal saline
- Remove the cannula, Lower arm on to a pillow and check tourniquet not leaking
- Perform the reduction of the fracture and obtain check x-ray,
- Watch for signs of toxicity. The cuff must be inflated for a minimum of **20 minutes and a maximum of 45 minutes**. If satisfied with the post reduction position of fracture, deflate the cuff observing the patient and monitor. Observe the patient and limb closely for signs of delayed toxicity until fully recovered. Check limb circulation prior to discharge. Arrange patient follow up and analgesia as appropriate

CHAPTER 11. INTER-HOSPITAL TRANSFERS

• TIMING OF TRANSFER

- The timing of a transfer will depend on the clinical needs of the patient, available staffing levels and time of day.
- Ideally transfers should take place during daylight hours but for reasons of clinical urgency this is not always possible.
- Transfers at night and at weekends are more hazardous due to team fatigue, the technical difficulties associated with driving or flying at night and, often, lower staffing levels of hospitals.
- The loss of a doctor or nurse from the transferring hospital may have a significant impact on a hospital with already reduced weekend or night-time staffing levels.
- Clinical urgency sometimes dictates that a transfer must take place as soon as possible.
- A typical example would be a traumatic brain injury requiring immediate surgical intervention such as the evacuation of an intracranial haematoma at a neurosurgical centre. In all cases a balance needs to be struck between optimising the patient prior to transfer and transferring promptly.
- In some cases, such as burns or trauma, it may not be possible to fully stabilise the patient prior to transfer.
- Senior staff at both the transferring and receiving hospitals must be involved in decision making about potential transfers and the timing of a transfer must be based on the risks and benefits to the patient of immediate or delayed transfer.

• CHOICE OF TRANSPORT

- Urgency, the distance to be travelled and the time of day all influence the choice of transport for transfer, as well as cost and availability.
- The principle modes of transport used include land ambulance, rotary aircraft (helicopters) and fixed wing aircraft (aeroplanes).
- Each mode of transport comes with its own unique advantages and disadvantages.
- **Land ambulances** are normally available 24 hours per day and can transport the patient door to door. However, they are often slower and less smooth than other forms of transport.
- **Air ambulances** are relatively fast and can provide virtually door to door transport. However they may be unable to fly in poor weather conditions or at night and internal space is limited, making patient care more challenging.
- **Larger helicopters**, such as those used by the military or civilian Search and Rescue organisations, provide more internal space, are able to fly at night and in most weather conditions. They are relatively fast and smooth. Due to the size of the craft, landing sites are more limited so an ambulance may be required to transport the patient to and from the landing site and hospital. These helicopters tend to be relatively dark inside, even in the daytime. They are also frequently cold inside, resulting in an increased risk of hypothermia to the patient.
- **Fixed wing aircraft** are often used for longer distance transfers or where transfer by road or helicopter is not practical such as international transfers. Depending on the size of the aircraft there can be greater space for patient care. Landing sites are even more limited and an ambulance transfer to and from the hospital and the airfield will be required. Loading and unloading of the patient stretcher can be challenging due to the combined weight of the patient and with weight and equipment.

• PREPARATION

- Preparation is the most important step in the transfer process. Even a simple issue like the loss of an intravenous cannula can represent a major problem.
- Replacing a cannula in a moving vehicle with limited access to the patient is extremely difficult and the problem becomes more serious if the cannula had been used for important infusions such as vasoactive or sedative agents.
- Prior to transfer, **all lines, endotracheal tubes, drains and monitoring** should be attached securely to the patient.
- **Back-up equipment and infusions** should be available, along with contact details for the transferring and receiving hospitals in case of any delays or problems during the journey. Doses of important drugs, for example mannitol, should be calculated prior to departure. If their use is likely, consider drawing these drugs up in advance.
- Many hospitals utilise checklists to ensure that nothing is missed during the preparation stage and the UK Intensive Care Society (ICS) publish such a system which most units use in the UK.
- By anticipating problems before they occur and having a plan in place to deal with them, transfers can be made substantially safer and less stressful.

• PACKAGING

- Packaging is how you physically prepare the patient for transfer.
- The aim is to minimise the risk of lines, tubes and equipment becoming disconnected and also to ensure the patient is kept safe during transport.
- The process of packaging should be considered layer by layer.
- The inner layer includes items physically attached to the patient such as ECG leads, pulse oximeters and endotracheal tubes.
- A systematic ABC approach should be used:
 - **The airway** should be secured, endotracheal tube length should be documented and position confirmed both clinically and radiologically prior to transfer.
 - Changing the patients' position may alter the position of the tube and this should be kept in mind if there is deterioration in ventilation or oxygenation en-route.
 - **ECG dots** should be covered with adhesive dressings to keep them attached to the patient and, where possible, ear probes should be used for pulse oximetry as these are more accessible and also more reliable if the patient's hands get cold.
 - Prior to departure **large bore IV access** should be obtained and secured with two adhesive cannula dressings.
 - **Arterial lines** should be sutured and covered with a large adhesive dressing. When siting central lines these should ideally be placed in the internal jugular vein for accessibility during transfer. However, delaying a time-critical transfer in order to site a central line is most often neither necessary nor best practice.
 - **Femoral lines** may be difficult to access once the patient is fully packaged and wrapped in blankets and if an air ambulance is the mode of transport they may be inaccessible due to the patients' position within the cabin. If this is the only available route, extension lines with an accessible three-way tap and flush bag should be in place so as to allow bolus administration of drugs.
- The next layer includes sheets and blankets.
- The final layer is the transport layer.
- This encompasses the scoops, stretchers, vacuum mattresses, splints and associated belts and securing devices.

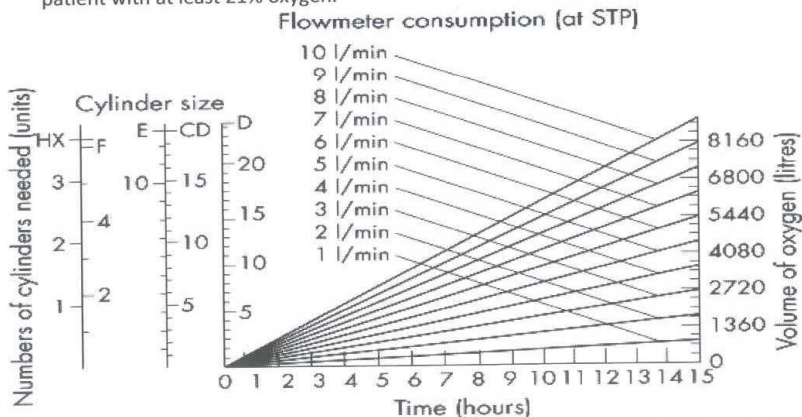
• EQUIPMENT

- The equipment for transfer should be familiar to the user, fully charged and in good condition. Spare batteries and additional backup equipment should be taken in case of failure or malfunction. Where possible equipment should be rationalised to save space and reduce the battery and mains power required.
- For example using boluses of the opiate drug being used rather than an infusion means you need one less pump to transfer the patient. One spare pump should be taken for every two that are used. An additional spare pump should be brought for each critical infusion, such as vasoactive agents. Non-invasive blood pressure cuffs use a lot of battery power so, where possible, arterial lines should be sited prior to transfer.
- As well as saving power they are also more accurate and allow continuous blood pressure monitoring. Immediately prior to leaving on a transfer it is important to think through potential complications and critical incidents that could occur, such as accidental extubation, and ensure that the equipment required to deal with these scenarios is readily available.
- Another important consideration is **how much oxygen the patient will require** for the journey. This should be accurately calculated with allowances made for delays during the journey and any additional oxygen requirements to drive the ventilator itself (known as the **"bias flow"**). The oxygen requirements for a straightforward transfer should be calculated and then this figure should be doubled. Always calculate using an **FiO₂ of 1.0** as this simplifies the calculations and offers the additional capacity to increase the oxygen delivery in case of deterioration en route.
- The total suggested oxygen that is required can be calculated using the equation shown below:

Litres of oxygen required = 2 x [(Minute Volume + Bias Flow) x duration of transfer in minutes]

Minute Volume = RR X TV (Resp. rate X Tidal Volume)

- Transfer equipment should always include a self-inflating bag-valve-mask. In the event of ventilator or oxygen failure this device will allow you to continue to ventilate the patient with at least 21% oxygen.



Oxygen cylinder duration plotted against number of oxygen cylinders required. Diagonal lines represent oxygen consumption at different flowmeter rates. Cylinder sizes: C, CD, E, F, HX.

• DURING TRANSFER

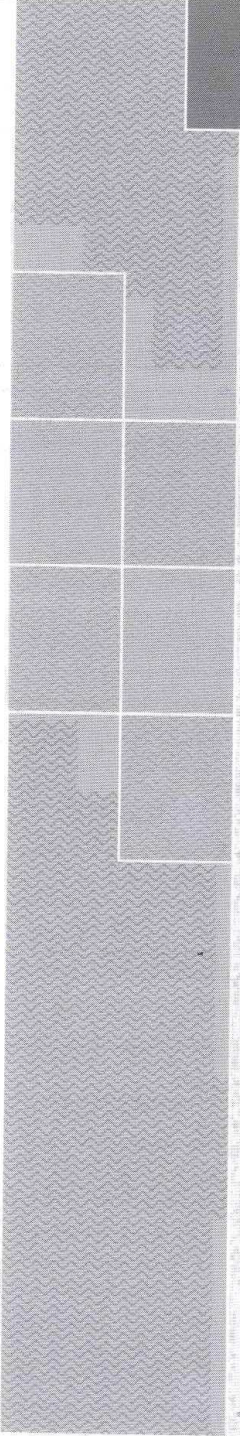
- Space can be limited during transfer. In a land ambulance, it is generally advised to sit to one side of the patient. However, this can make access to the patients' opposite side difficult and this should be considered when positioning cannulas.
- In an air ambulance, access to the patient's lower body is often difficult due to the tail portion of the cabin, so the use of femoral lines should be avoided.
- In all forms of transport, noise, vibration and motion make patient monitoring more difficult. Visual checks of the patient, monitoring and equipment must be performed at regular intervals. To facilitate this attach pumps, ventilators and monitors to the trolley or equipment bar (if available) so they are clearly visible. The ability to auscultate, palpate and percuss may also be severely reduced by noise and vibration.
- This makes reliable monitoring such as arterial lines, pulse oximetry and capnography very important. In the ambulance or aircraft additional equipment and drug bags should be securely stored to prevent them moving and becoming an obstacle or trip hazard. During transfer, aim to complete any interventions or examinations of the patient whilst seated, as this reduces the chance of injury. If it is necessary to move, aim to do so when the vehicle is steady and kneel rather than stand, as this is a more stable position. All forms of transport can cause motion sickness and fatigue which may affect both patient and crew. Motion sickness may develop when focus is being directed at monitoring equipment and the patient rather than external visual cues and added to by vibration from engines or rotors. When travelling by air, flicker from rotor blades or strobe lights used for navigation can be a source of fatigue and in susceptible individuals may even lead to seizures. It is important to be aware of the impact that motion sickness may have on your performance. If motion sickness becomes an issue looking out of the windows of the ambulance regularly and trying to keep the environment cool may help. Antiemetics may be necessary.

• DOCUMENTATION

- Documentation is crucial during transfer; it provides a legal record of the patient's condition, the care provided and any complications encountered during the journey.
- During transfer the patient's vital signs and details of any medications or infusions administered should be recorded. A set of observations prior to transfer should be documented so that trends can be assessed. By providing a clear and concise written record of the patient's condition and current status, the chance of anything being missed when handing over to the receiving hospital is reduced.
- A photocopy of the patient's medical and nursing records should also be given to the receiving team and any clinical imaging should be made available. Copies of the transfer records should be kept by both the receiving and transferring hospital.

• SAFETY

- Safety is important during transfers and this applies not only to the patient, but to all the staff transferring the patient. The first aspect to be considered is appropriate clothing for transfer. Transfer clothing should be warm and, for safety, a hi-visibility jacket should be worn along with sturdy shoes. Ideally coveralls should be worn and if travelling by air a helmet may also be required. Safety on transfer also involves using common sense and trusting your instincts; if you feel a situation is unsafe discuss it with the ambulance or aircrew. Despite all steps being taken, accidents do still happen and it is important that you protect yourself by taking out personal accident insurance. Membership of some professional bodies such as the UK ICS includes such cover.



INTENSIVE CARE MEDICINE

2 QUESTIONS

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CHAPTER 1. ARTERIAL BLOOD GASES (ABG)

I. NORMAL ABG VALUES

- The first portion of an ABG assay usually reports pH, pO_2 and pCO_2 .
- The partial pressure of O_2 and CO_2 is expressed as either kilopascals (kPa) or millimetres of mercury (mm Hg).
- Normal values (on inspired room air) are as follows:

pH	7.350-7.450	
	kPa	mm Hg
pCO_2	4.67-6.00	35-45
pO_2	10.67-13.33	80-100
Bicarbonate	23-28 mmol/L	
Base excess	-2 to +2 mmol/L	

- **BASE EXCESS / BASE DEFICIT**
 - Calculation of the base excess or deficit is a way of quantifying HCO_3^- .
 - Base excess is the quantity of base (HCO_3^- , in mEq/L) that is above or below the normal range of buffer base in the body (22 -28 mEq/L).
 - This cannot be calculated from PCO_2 and pH as the hemoglobin also contributes to the buffer base.
 - One can use the **Siggaard-Andersen Nomogram** to estimate base excess or deficit.
 - **Severe metabolic acidosis** is associated with a base deficit of -10 mEq/L
 - A **positive number** is called a **base excess** and indicates a **metabolic alkalosis**.
 - A **negative number** is called a **base deficit** and indicates a **metabolic acidosis**.

ACID-BASE DISTURBANCES CAN BE OF EITHER:

- **Respiratory origin** where the disturbance is primarily of CO_2 exchange
- **Metabolic origin** where the disturbance is due to bicarbonate
- if the pH moves towards normal, this is termed **compensation**.
- **Correction** is when normal pH is restored.
- The next compensatory change in pH occurs by **altering respiratory rate** and therefore blood pCO_2 .

- **AN INCREASE IN CO_2 REDUCES THE PH:**



↑ CO_2 shifts the equation to the right → $H_2CO_3 \rightarrow \uparrow H^+$ i.e. ↓pH

↓ CO_2 shifts the equation to the left → ↓ H^+ i.e. ↑pH

- The final compensatory change is renal handling (i.e. excretion) of acid (and subsequent reabsorption of HCO_3^-).

II. UNCOMPENSATED ACID-BASE DISORDERS**1. METABOLIC ACIDOSIS****↓pH, ↓HCO₃⁻**

- Classification of a metabolic acidosis depends on the anion gap – the difference between the major plasma cations (Na⁺ and K⁺) and anions (Cl⁻ and HCO₃⁻):

$$\text{Anion gap} = (\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$$

- A normal anion gap is in the range **9-14 mmol/l**.
- Calculating the anion gap often helps **identify the cause of the acidosis**.
 - **HAGMA**: High Anion Gap Metabolic Acidosis.
 - A raised anion gap can be due to *excess acid production or ingestion* contributing extra H⁺: **CAT MUDPILERS**
 - **NAGMA**: Normal Anion Gap Metabolic Acidosis.
 - In a normal anion gap acidosis, *bicarbonate is lost from the gut or the kidneys* and there is a raised chloride, which compensates for the extra cations, thus keeping the gap normal. This occurs as a result of reabsorption of sodium chloride via the kidneys: **PARAMEDIC**

METABOLIC ACIDOSIS AND ANION GAP

HAGMA		NAGMA	
C	CO/Cyanide	P	K ⁺ sparing diuretics (Spironolactone)
A	Alcohol/Aminoglycosides	A	Acetazolamide
T	Toluene	R	Rhabdomyolysis / RTA
		A	Alimentation feeding
M	Methanol	M	Mineral Acids
U	Urea	E	Enterostomy
D	DKA (and AKA)	D	Diarrhoea
P	Paracetamol/Paraldehyde/ Phenformin	I	Intestinal fistula
I	Iron / Isoniazid	C	Cholestyramine
L	Lactic acidosis	Or ABCD : Addison crisis, Bicarbonate loss (GIT, Renal/RTA), Chloride excess and Drugs (Acids, Spironolactone, Acetazolamide, cholestyramine)	
E	Ethanol/Ethylene glycol		
S	Salicylate OD/Solvents /Starvation		

2. METABOLIC ALKALOSIS**↑pH, ↑HCO₃⁻**

- **CAUSES OF METABOLIC ALKALOSIS:**
 - Direct loss of H⁺ in gastric secretion: Vomiting, Nasogastric suction
 - Conn's syndrome
 - Hypokalaemia
 - Excess alkali
 - IV Bicarbonate administration in large amounts
 - Ingestion of antacids

3. RESPIRATORY ACIDOSIS

↓pH, ↑pCO₂

- There are two types of respiratory failure essentially distinguished by the levels of CO₂ within the blood and the main driver for ventilation.

A. TYPE 1 RESPIRATORY FAILURE: OXYGENATION FAILURE

- In Type 1 Respiratory Failure (where patients are typically hypoxic but not hypercapnic), the hypercapnic drive is more effective than the hypoxic.
- Patients can be given supplemental O₂ therapy.

B. TYPE 2 RESPIRATORY FAILURE: VENTILATION FAILURE

- Patients are typically hypoxic and hypercapnic.
- If acutely unwell patients are given supplemental oxygen in these circumstances, the hypoxic stimulus is no longer present and ventilation is suppressed; CO₂ rises further and the patient becomes increasingly acidotic.
- The lack of O₂ causes anaerobic respiration at a cellular level, producing lactic acid, worsening the acidosis.

Hypoxaemic (Type I)	Hypercapnic (Type II)
<ul style="list-style-type: none"> • Pneumonia • ARDS • Pulmonary fibrosis • Asthma • COPD • Pneumothorax • PE • Obesity • Pulmonary hypertension 	<ul style="list-style-type: none"> • COPD/ Severe Asthma • Drug overdose (Opiates, Benzodiazepines) • CNS injury (CVA, SCI) • Primary muscle disorders (Duchenne muscular dystrophy) • Neuromuscular junction disorders (Myasthenia gravis) • Anatomical chest deformities (Kyphoscoliosis, Flail chest) • Obesity hypo-ventilatory (Pickwickian) syndrome

4. RESPIRATORY ALKALOSIS

↑pH, ↓pCO₂

- In **respiratory alkalosis**, there is low pCO₂ and a consequent high pH as a result of the equation **moving to the left** and lowering H⁺.
- **CAUSES OF RESPIRATORY ALKALOSIS:**
 - Anxiety, Pain, Fever
 - Hypoxia, CHF, Hypotension
 - P.E., Pneumonia, Sepsis
 - Drug use: Salicylates, Catecholamines, Progesterone
 - Pregnancy
 - Hepatic encephalopathy, Liver failure
 - Mechanical ventilation
 - Hypothyroidism
 - High altitude

III. COMPENSATION OF ACID-BASE DISORDERS

- Compensatory mechanisms restore pH towards normal by altering $p\text{CO}_2$ and HCO_3^- .

1. COMPENSATION OF METABOLIC ACIDOSIS

- o The lowered pH acts on peripheral chemoreceptors to stimulate the ventilation. *Respiratory rate increases and $p\text{CO}_2$ falls: $\uparrow\text{RR}$, $\downarrow\text{Pco}_2$*
 $\downarrow\text{CO}_2 + \text{H}_2\text{O} \leftarrow \text{H}_2\text{CO}_3 \leftarrow \text{H}^+ + \text{HCO}_3^-$
- o $[\text{H}^+]$ therefore falls, as does $[\text{HCO}_3^-]$.
- o There is also increased reabsorption of HCO_3^- and increased excretion of H^+ from the kidneys but this is not instant.

2. COMPENSATION OF METABOLIC ALKALOSIS

- o Conversely, in metabolic alkalosis, the higher pH acts on the chemoreceptors to reduce ventilation and increase $p\text{CO}_2$: $\downarrow\text{RR}$, $\uparrow\text{Pco}_2$
 $\uparrow\text{CO}_2 + \text{H}_2\text{O} \leftarrow \text{H}_2\text{CO}_3 \leftarrow \text{H}^+ + \text{HCO}_3^-$
- o The renal response is then to decrease HCO_3^- reabsorption and decrease H^+ excretion. This usually occurs fairly quickly, but if the alkalosis is caused by vomiting, resulting in dehydration, the overriding renal response is to increase Na^+ and HCO_3^- reabsorption.
- o Therefore, effective rehydration will help to more rapidly correct the alkalosis.

3. COMPENSATION OF RESPIRATORY ACIDOSIS

- o The problem here is within the ventilatory system, with the kidneys acting to compensate which can take a significant length of time (**up to two days**).
- o The $[\text{H}^+]$ is raised, thus the rate of H^+ secretion is also increased.
- o This results in **increased HCO_3^- reabsorption**, despite HCO_3^- already being higher as a result of the equation shifting to the right:
 $\uparrow\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \uparrow\text{H}^+ + \uparrow\text{HCO}_3^-$
- o Although the secretion of H^+ brings the pH closer to normal, the pH will not be restored to normal without correction of the underlying respiratory disorder.

4. COMPENSATION OF RESPIRATORY ALKALOSIS

- o In respiratory alkalosis, the $[\text{H}^+]$ decreases due to a primary reduction in $p\text{CO}_2$. There is therefore less H^+ in the renal tubules and reduced H^+ secretion.
- o As a consequence, **less HCO_3^- is reabsorbed causing a further fall in $[\text{HCO}_3^-]$** .
- o To restore $p\text{CO}_2$ and HCO_3^- completely to normal, the primary ventilatory problem must be corrected (i.e. The respiratory rate must reduce).

IV. MIXED ACID-BASE PICTURE

- A mixed acid-base disturbance is where there is **more than one primary disorder at a time**. This often occurs in acutely unwell patients.
NB: it is impossible to have more than one respiratory disorder in a mixed picture. i.e. a metabolic acidosis and alkalosis can co-exist, but not a respiratory acidosis and alkalosis.
- When considering all mixed disturbances, the clinical picture will usually indicate the underlying problem – the blood gas results should always be put in a clinical context.

1. METABOLIC ACIDOSIS AND RESPIRATORY ALKALOSIS

- **MACRAL** occurs in:
 - Septic shock
 - Sepsis and renal failure
 - Salicylate overdose
 - CCF and renal failure
 - Early cardiopulmonary arrest
- It can be difficult to distinguish this mixed picture from a compensated primary metabolic acidosis or respiratory alkalosis.
- **Diagnostic criteria:**
 - The clinical picture
 - Very low HCO_3^- : compensation from a respiratory alkalosis rarely causes a fall in HCO_3^- below 18.
 - Very low CO_2 : if this is very low, again, it is unlikely to be from a compensatory response alone
- For example, in a patient with septic shock, secondary to a urinary tract infection:

pH	7.37
pCO ₂	3.8 kPa
pO ₂	11.6 kPa
bicarbonate	12.4 mmol/L
lactate	4.5 mmol/L

- The patient is hyperventilating, attempting to increase the amount of oxygen to underperfused tissues. The CO_2 is therefore blown off, but bicarbonate is very low, indicating a metabolic component.
- The lactate is high reflecting sepsis and despite all this, the pH is **surprisingly normal**. Remember not to be falsely reassured by the normal pH – the clinical picture will reveal an unwell patient.

2. METABOLIC ALKALOSIS AND RESPIRATORY ACIDOSIS

- **MALRAC** occurs in:
 - Diuretic therapy (with low K^+) and COPD (poor gas exchange with retention of CO_2)
 - Vomiting (loss of H^+) and COPD
- **Diagnostic criteria:**
 - Clinical picture
 - Very High HCO_3^- : $\text{HCO}_3^- > 45 \text{ mmol/L}$, it is much more likely to be a mixed disorder.
 - High CO_2
- For example, in a patient on long term thiazide medication for hypertension who presents with an acute exacerbation of COPD:

pH	7.36
pCO ₂	7.8 kPa
pO ₂	7.8 kPa
Bicarbonate	48 mmol/L
Potassium	3.2 mmol/L

- It would be easy to be reassured by this patient's normal pH, but the low potassium and acute presentation of the breathing difficulty should point to this being a more complicated picture.
- The bicarbonate is **unusually high**, even for metabolic compensation of a respiratory disorder. The clinical condition of the patient should be treated rather than simply relying on a normal pH.

3. METABOLIC ALKALOSIS AND RESPIRATORY ALKALOSIS

- **MALRAL** occurs in:
 - Diuretic therapy (low potassium) and pneumonia
 - Vomiting (H^+ loss) and congestive cardiac failure (hyperventilation)

- **Diagnostic criteria**
 - *Clinical picture*
 - HCO_3^- raised
 - pCO_2 will be reduced
- For example, in a patient who has been **vomiting profusely** and is, as a result, highly anxious with raised respiratory rate:
 - pH **7.53**
 - pCO_2 **2.7 kPa**
 - pO_2 **10.9 kPa**
 - bicarbonate **34 mmol/L**

◦ *The clinical picture needs to be considered*
 ◦ *This patient is alkalotic with a low CO_2 , indicating a respiratory component, but the bicarbonate is high, rather than normal or low.*

4. METABOLIC ACIDOSIS AND METABOLIC ALKALOSIS

- **MACMAL** Occurs in:
 - *The combination of a raised anion gap metabolic acidosis (e.g. Uraemia, ketoacidosis, lactic acidosis) and vomiting (loss of H^+)*
 - *The combination of a raised anion gap metabolic acidosis and diuretic therapy (with low K^+)*
 - *Lactic or ketoacidosis and bicarbonate therapy*
- For example, in a patient with diabetic ketoacidosis secondary to infection and vomiting:
 - pH **7.37**
 - pCO_2 **3.4 kPa**
 - pO_2 **10.2 kPa**
 - bicarbonate **6.5 mmol/L**

◦ *A patient with ongoing ketoacidosis who has compensated to the extent that the bicarbonate is only 6.5 is unlikely to have fully restored their pH to normal levels.*
 ◦ *This is a falsely reassuring picture and the clinical condition should be considered, or the patient could be undertreated. Therefore if the patient is also vomiting, consider direct loss of $[\text{H}^+]$.*

V. OTHER RESULTS ON THE ARTERIAL BLOOD GAS

- Other results are detailed below.
 - **Potassium:** when low, this indicates a possible **Metabolic Alkalosis**.
 - **Chloride:** this also has a bearing on metabolic acid-base disorders and is required for calculating the anion gap
 - **Lactate:** immensely important in the diagnosis of **Sepsis** and **global hypoperfusion**.
 - A lactate **>4 mmol/l** in the presence of suggested infection would initiate Early Goal Directed Therapy.
 - Remember, \uparrow lactate in the presence of abdominal pain suggests **Ischaemic bowel**
 - **Glucose:** remember raised glucose with acidosis can indicate **ketoacidosis**. Glucose is also an important target in the Surviving Sepsis Campaign: it should be maintained above the lower limit of normal, **but less than 8.3 mmol/l**.
 - **Haemoglobin:** if the Hb is low, there is less O_2 carrying capacity within the blood
 - **Carboxyhaemoglobin:** CO binds to Hb to form carboxyhaemoglobin (COHb);
 - It binds **230-270 times more strongly than O_2** and causes a leftward shift in the oxyhaemoglobin dissociation curve, so less oxygen is available to hypoxic tissues.
 - The main early symptom is headache which occurs when levels reach around **10%**.
 - But when levels reach **50-70%**, seizures and death can result.
 - When breathing air, CO has a **half-life of 3-4 hours**, but only **30-90 minutes** when breathing 100% O_2 . Hyperbaric oxygen reduces this further.

CHAPTER 2. ABNORMAL BLOOD GLUCOSE

I. HYPOGLYCAEMIA

- A 50 year-old man presented to the emergency department confused and ataxic with slurred speech. He was mildly tremulous, sweating and mildly tachycardic and hypertensive. Although he appeared drunk, his breath alcohol test was negative.
- He had no known comorbidities and did not take any regular medications. His fingerprick blood glucose reading was **1.6 mmol/L**.

Q1. Is the presentation in keeping with hypoglycemia?

- Yes.
 - He has a combination of autonomic symptoms (e.g. sweating, tachycardia, tremor) and neuroglycopenic symptoms (e.g. confusion, ataxia, dysarthria).
 - Hypoglycemia accounts for over 5% of ED presentations of 'altered mental state'.
 - Hypoglycemia can cause focal neurological signs — don't miss hypoglycemia masquerading as stroke!
 - The 'drunk' patient may be hypoglycemic — even when they are actually intoxicated with alcohol...

Q2. What is Whipple's triad?

- **Whipple's triad** confirms the diagnosis of clinically significant hypoglycemia. It consists of:
 - The presence of symptoms consistent with hypoglycemia
 - A low serum glucose level
 - Resolution of the symptoms and signs of hypoglycemia with the administration of glucose

Q3. What are the causes of hypoglycemia? "DIABETES EXPLAINS H"

- **Diabetes:**
 - The vast majority of ED hypoglycemia presentations involve patients with diabetes that are taking **insulin or oral hypoglycemic drugs**.
 - Factors that contribute to hypoglycemia include *missed meals, incorrect medication dosage or administration, intercurrent illnesses, alcohol consumption, increased exercise and deteriorating renal function*.
- **Fasting hypoglycaemia: EXPLAINS H**
 - **Exogenous drugs:** Insulin, Oral Hypoglycemics, Quinine, Chloroquine, Beta-Blocker Overdose, Valproate Overdose, Salicylate Overdose, Pentamidine.
 - Pituitary insufficiency
 - Liver disease: Hepatocellular Cancer, Hepatitis and rare genetic defects.
 - Addison's disease
 - Islet cell tumours: Insulinomas
 - Immune hypoglycaemia: e.g. anti-insulin receptor antibodies in Hodgkin's disease or anti-insulin antibodies that release insulin when insulin levels are relatively low;
 - Infection: e.g. Severe Sepsis, Malaria
 - Non-pancreatic neoplasms: fibromas, sarcomas, mesotheliomas, and small cell carcinomas that produce IGF-2; extensive metastases that overwhelm the body's ability to produce glucose;
 - **Nesidioblastosis or Noninsulinoma pancreatogenous hypoglycemia (NIPH) syndrome:** islet cell hyperplasia, which can be congenital or acquired, e.g. post-gastric surgery

- Starvation and Malnutrition
- Hypothyroidism (myxoedema coma)
- **Post-prandial hypoglycaemia:**
 - Can also occur due to a rapid surge of insulin ('late dumping') following rapid entry of food into the small intestine.
 - This may occur after gastric surgery, for instance.
- **Pseudohypoglycemia:**
 - Leukocytosis, Thrombocytosis, or Erythrocytosis can cause excess consumption of glucose in the collection vial while the sample awaits testing.

Q4. What is the emergency treatment of hypoglycemia?

- **In the awake, cooperative patient:**
 - **Oral intake of simple carbohydrates** (e.g. A sugary drink, jam, sugar lumps, barley sugars, etc) followed by a sandwich or biscuits.
- **In the uncooperative or unconscious patient**, parenteral therapy is needed:
 - **50 mL 50% Glucose** (or 5 mL/kg of 10% glucose in small children) — flush with saline as it is hypertonic and can cause **phlebitis**.
 - **Glucagon 1mg IM/SC or IV**. This may be administered by family members or by paramedics at the scene when IV access is difficult. It is inappropriate in settings where liver glycogen stores are depleted (e.g. liver failure or chronic alcoholism).
 - Full neurological recovery is usually rapid, and is expected within about 20 minutes — otherwise suspect a complication (e.g. stroke) or an alternative coexisting diagnosis.
- **Octreotide infusion** in the case of sulfonylurea poisoning.

Q5. What is the role of thiamine in the treatment of hypoglycemia?

- It is traditionally advocated that thiamine 100mg IV be given prior to administering a bolus of glucose to the patient with altered mental status. This comes from the concern that **Wernicke's encephalopathy** may be precipitated or exacerbated by the glucose load in the absence of thiamine administration.
- *"Thiamine is a cofactor for several essential enzymes in the Krebs cycle and the pentose phosphate pathway, including α -ketoglutarate dehydrogenase, pyruvate dehydrogenase, and transketolase. Because thiamine-dependent enzymes play an important role in cerebral energy use, deficiency may initiate tissue injury by inhibiting metabolism in brain regions with high metabolic requirements and high thiamine turnover."* — Donino et al, 2007.
- *The concern is that an excessive carbohydrate load will lead to the build up of toxic metabolites when the activity of these enzymes is reduced because of thiamine deficiency. However, there appears to be no instances of a single bolus of glucose precipitating Wernicke's encephalopathy, although prolonged carbohydrate administration (e.g. from total parenteral nutrition) without thiamine supplementation certainly can.*
- **The bottom line is:**
 - Never delay the correction of hypoglycemia because of an irrational desire to administer thiamine first.
 - By all means give thiamine (and magnesium, another cofactor, while you're at it) — **especially to the alcoholic or malnourished patient** — it is safe and is an effective treatment for Wernicke's encephalopathy.

Q6. What investigations are required in the patient presenting with hypoglycemia?

- In the otherwise well patient with diabetes who simply missed a meal, it might be that no further investigations are needed.
- Investigations should be appropriate to the clinical situation and aim to identify the causes and complications of the hypoglycemic episode.
- Useful investigations may include: Glucose, Insulin, Beta-Hydroxybutyrate, C-peptide
 - **High/Normal insulin with no excess ketones** is consistent with insulinoma, sulfonylureas, insulin administration and insulin autoantibodies.
 - C-peptide is absent if exogenous insulin is administered.
 - **Low insulin with no excess ketones** is consistent with anti-insulin receptor antibodies and non-pancreatic neoplasms.
 - Low insulin with high ketones is consistent with ethanol-induced hypoglycemia, pituitary and adrenal failure.
- **Consider other causes**, such as:
 - Septic screen, thick and thin films for malaria, or malaria antigen tests.
 - LFTs and INR (liver disease)
 - UEC, 24h creatinine clearance, renal imaging (renal failure)
 - **Endocrine tests:**
 - Cortisol (adrenal insufficiency)
 - GH response to hypoglycemia stimulation test, synACTHen test (pituitary failure)
 - TFTs (hypothyroidism)
 - Monitored prolonged fasting for recurrent hypoglycemia (especially if investigations were not obtained on the presenting episode of hypoglycemia)
 - Tests for insulinomas — absence of C-peptide suppression following insulin administration, pancreatic arterial calcium-stimulation and vein sampling.
 - Autoantibodies — anti-insulin receptor and anti-insulin antibodies
 - Tumour imaging (e.g. MRI)
 - Drug levels if occult drug administration is suspected (e.g. sulfonylureas).

II. DIABETIC KETOACIDOSIS IN ADULT

Introduction

- **DKA is defined as:**
 - Ketonaemia > 3.0 mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
 - Blood glucose > 11.0 mmol/L or known diabetes mellitus
 - Serum venous bicarbonate of < 15.0 mmol/L and/or venous pH < 7.3

PATHOPHYSIOLOGY – DKA

- **Insulin actions**

INHIBITS	INDUCES
Gluconeogenesis	Glucose uptake
Glycogenolysis	Glycolysis
Ketogenesis	Glycogen synthesis
Lipolysis	Protein synthesis
Proteinolysis	Uptake of ions, especially potassium

- **Fluid and electrolyte losses in typical DKA**

Water ml/Kg	100
Sodium mmol/Kg	7-10
Potassium mmol/Kg	3-5
Chloride mmol/Kg	3-5
Magnesium mmol/Kg	1-2
Calcium mmol/Kg	1-2
Phosphate mmol/Kg	1-1.5

Clinical Assessment and Risk Stratification

- The annual incidence of DKA is between 1-5% of patients with **type I diabetes**.
- Patients who present in DKA may complain of polyuria, polydipsia, weakness, nausea, vomiting (50-80%), coffee-ground haematemesis (25% of vomiting patients) and abdominal pain (30%).
- Physical findings are, *dry mucous membranes, tachycardia, hypotension, alteration in mental state, sweet smell to the breath and Kussmaul's respirations*.
- In a patient who presents with **abdominal pain and vomiting** the diagnosis of DKA can easily be missed

Investigations

- **Initial bedside tests include:**
 - Capillary **blood glucose**
 - **Blood gases** to determine pH, bicarbonate and potassium
 - **Urine dipstick** for ketones and urinalysis
 - **ECG** to investigate the possibility of a myocardial infarction, which may be silent
- There is no need to take arterial blood routinely in suspected DKA.
- Venous blood can be used, as the mean difference between arterial and venous pH is 0.03. Arterial sampling should only be undertaken if there is a concern that there is respiratory failure.

Management of DKA in the ED

Emergency Department

Adult Diabetic Keto-Acidosis

Newham University Hospital 
NHS Trust

Initial management to be completed within 1 hour of arrival

Establish Diagnosis

Known diabetes or high Blood Glucose or BM: 11 or higher
+
Ketones: Urinalysis 2+ ketones or more
+
Acidosis: Blood Gas (venous or arterial):
pH 7.30 or lower or Bicarb 15 or lower or BE worse than -10

All patients with BM 11 or more must have a urinalysis
DKA may be the first presentation of diabetes

Pitfalls:
Euglycaemic DKA
Non-ketotic DKA

Move to Resus

Inform ED middle grade or consultant
Inform Medical & O&G registrar immediately if patient is pregnant
(contact medical registrar +/- ITU middle grade to assist at any time)

Investigations & Monitoring

FBC, U&E, blood glucose (before treatment)
Blood cultures
Consider urinary catheter
Urine culture
Attach cardiac & sats monitoring
Obtain iv access (minimum 18 G cannula)
ECG
CXR only if indicated

If systolic BP < 90mmHg

Consider stat dose 500mls 0.9% Saline
Call for senior help
Caution: young adults 18-24 yrs, pregnancy, elderly, heart failure
(refer to Trust policy)

Treatment

1. Fluids:

BM < 14 Give 10% Glucose AND 0.9% Saline

1 L 10% Glucose over 8 hours (125 mL / hr)
1 L 0.9% Saline as per saline regime

Run through 2 separate lines at the **SAME** time

If hypoglycaemia occurs **INCREASE** rate of glucose infusion to permit insulin administration

BM 14 or more Give 0.9% Saline

1 L over 1 hour
1 L over 2 hours
1 L over 2 hours
1 L over 4 hours
1 L over 4 hours

Continue 0.9% Saline infusion until volume replete
Add KCl as per guideline below **EXCEPT** to 1st bag

2. Insulin (whatever BM):

No stat dose

Start fixed rate insulin infusion of 50 units actrapid in 50 ml 0.9% saline ie 1 unit per mL solution
Run at 0.1 units / kg / hr (may need to estimate weight)

Aim: Fall in blood glucose by 3 mmol / hr OR rise in HCO₃ by 3 mmol / hr.

Consider increasing rate of insulin infusion by 1 unit / hr to achieve this

3. Potassium - Do not add potassium to first bag of fluid

Await results before adding K⁺ to iv fluids

Replace at rate of **no greater than 20 mmols / hour** via peripheral line

K⁺ > 5.5 nil

K⁺ 3.5-5.5 add 40 mmol in 1 litre 0.9% Saline.

K⁺ < 3.5 may require additional KCl (seek expert help)

4. BM

Check hourly

When BM < 14, patient needs to have 10% glucose (1L over 8 hours) as well as 0.9% Saline to avoid hypoglycaemia whilst on insulin infusion. Prescribe appropriately on drug chart

Recheck blood gas, U&E, blood glucose, CK within 2 hours

DKA COMPLICATIONS

- There are potential complications of the treatment of the patient with DKA:
 - o **Hypoglycaemia**
 - This is caused by the administration of insulin.
 - Hourly monitoring of blood sugar concentration is needed to avoid this complication.
 - o **Hypokalaemia**
 - Hypokalaemia is a common complication, exacerbated by starting insulin in the face of hypokalaemia, inadequate potassium replacement or by the use of sodium bicarbonate.
 - Hypokalaemia can lead to muscle cramping or weakness, nausea or vomiting, polyuria, polydipsia, psychosis, delirium, hallucinations and importantly cardiac arrhythmias and cardiac arrest.
 - o **Cerebral oedema**
 - Fortunately, cerebral oedema is very rare in adults. Multiple factors in the treatment may contribute to cerebral oedema; these include osmotically active particles in the intra-cellular space driven by the insulin and rapid changes in sodium concentrations.
 - This risk can be minimised by slow correction of hyperglycaemia and avoiding overzealous fluid replacement.
 - o **Acute respiratory distress syndrome (ARDS)**
 - The partial pressure of oxygen steadily decreases during treatment to low levels.
 - This is believed to be due to interstitial oedema and reduced lung compliance.
 - The mechanism is similar to that causing cerebral oedema.
 - o **Hyperchloraemic metabolic acidosis**
 - This is common, due to the loss of substrates in the urine that are necessary for bicarbonate regeneration, the large concentration of chloride infused in intravenous fluids and the shift of fluids if sodium bicarbonate is used.
 - The acidosis normally corrects in the subsequent 24 to 48 hours through increased renal excretion.
 - However, the persistent base deficit can catch out the unwary.

III. HYPEROSMOLAR HYPERGLYCAEMIC STATE

INTRODUCTION

- Hyperosmolar Hyperglycaemic State (HHS) or Hyperosmolar non-ketotic acidosis (HONK) is a condition that can occur in people with **very high blood glucose levels**, caused by a severe lack of insulin.
- HHS usually presents in older patients with type 2 DM and carries a higher mortality than DKA, estimated at approximately 10-20%.
- HHS is characterized by hyperglycemia, hyperosmolarity, and dehydration **without significant ketoacidosis**.
- Infection is the most common preceding illness, but many other conditions can cause altered mentation, dehydration, or both.
- Once HHS has developed, it may be difficult to differentiate it from the antecedent illness. The concomitant illness may not be identifiable.
- HHS has also been reported in patients **with type 1 DM**, in whom DKA is more common.
- Most patients present with severe dehydration and focal or global neurologic deficits.
- In as many as one third of cases, the clinical features of HHS and DKA overlap and are observed simultaneously (overlap cases); this suggests that these 2 states of uncontrolled DM differ only *with respect to the magnitude of dehydration and the severity of acidosis*.

DIAGNOSTIC CRITERIA

- *Hyperglycaemia: Glucose > 30 mmol/l*
- *Total osmolality: > 340mOsm/kg*
- *Serum bicarbonate: >15mmol/l*
- *Urinary ketones: < 1+ plus*

MANAGEMENT

- Oxygen.
- +/- CVP monitoring to guide fluid replacement.
- NG tube if ↓ level of consciousness.
- Urinary catheter.
- Prophylactic heparin (Innohep), if no contraindications.

CHAPTER 3. VASOACTIVE DRUGS

I. RECEPTORS

1. ADRENERGIC RECEPTORS

<i>Adrenoreceptors</i>	α_1	<ul style="list-style-type: none"> • Vasoconstriction • Increased Peripheral Resistance • Increased BP • Mydriasis • Increased closure of internal sphincter of bladder
	α_2	Inhibition of: <ul style="list-style-type: none"> • Norepinephrine release • Ach release • Insulin release
	β_1	<ul style="list-style-type: none"> • Increased heart rate. • Increased myocardia contractility • Increased release of renin • Increased lipolysis • Increased platelet aggregation
	β_2	<ul style="list-style-type: none"> • Vasodilation • Bronchodilation • Slightly decreased Peripheral resistance • Increased muscles and liver glycogenolysis • Increased release glucagon • Relax uterine smooth muscles
	β_3	<ul style="list-style-type: none"> • Lipolysis

2. DOPAMINERGIC RECEPTORS (DA):

- These receptors exist mainly in the splanchnic circulation and **lead to vasodilation**.
- Renal dopaminergic receptors will lead to an **increase in renal blood flow**.

3. VASOPRESSIN RECEPTORS (V):

- These receptors are separate from the adrenergic system.
- There are three subtypes of this receptor, but V1 receptors located on vascular smooth muscle cells, producing arterial vasoconstriction when activated, are the most important subtype.
- V1 receptors also exist on platelets, hepatocytes, and the myometrium, with actions that vary based on location.

II. INOTROPE AND VASOACTIVE AGENTS

1. INOTROPES:

- Agents that **increase myocardial contractility** or inotropy (**β_1 effect**)
- e.g. **Dobutamine**, Adrenaline, Isoprenaline, Ephedrine
 - **Positive inotropes** increase the force of contraction of the heart.
 - **Negative inotropes** weaken the force of contraction of the heart.

2. VASOPRESSORS:

- Agents that **cause vasoconstriction** leading to **increased** systemic and/or pulmonary vascular resistance (**\uparrow SVR, PVR/ α_1 effect**)
- e.g. **Noradrenaline**, Vasopressin, Metaraminol, Methylene Blue

3. INODILATORS:

- Agents with **inotropic effects that also cause vasodilation** leading to **decreased** systemic and/or pulmonary vascular resistance (**\downarrow SVR, PVR**)
- e.g. **Milrinone**, Levosimendan
- Some agents (Dopamine) don't fit any of these categories.

MECHANISM OF ACTION

- The main mechanism of action for most inotropes involves increasing intracellular calcium, either by increasing influx to the cell during the action potential or increasing release from the sarcoplasmic reticulum. The main receptor in the cardiac muscle that affects the rate and force of contraction is the β_1 receptor.
- Binding to β_1 receptors results in increased calcium entry into the cell via the opening of L-type calcium channels and release of intracellular calcium from the sarcoplasmic reticulum. More calcium is available to bind with troponin-C, thereby enhancing myocardial contractility. Choice of inotrope will depend on factors such as the patient's underlying disease state and the clinician's preference.

INDICATIONS

- Most commonly used inotropes are the catecholamines; These can be:
 - **Endogenous:** Adrenaline, Noradrenaline
 - **Synthetic:** Dobutamine, Isoprenaline.
- These medicines act on the sympathetic nervous system.
- Most commonly their cardiac effects are attributed to stimulation of alpha and beta adrenergic receptors (specifically α_1 , β_1 , and β_2).
- Most catecholamines have a **short half-life** (about 2 minutes) and steady-state blood concentrations are reached within 10 minutes.
- They are therefore usually given by continuous infusion.
- Inotropes are indicated in acute conditions where there is low cardiac output (CO), such as **cardiogenic shock** following myocardial infarction, **acute decompensated heart failure** and low CO states after cardiac surgery.
- *It is important to optimise preload by correcting fluid balance before starting inotropes, since there is little point in increasing the contractility of the heart if its chambers are not filled optimally. Central venous pressure (CVP) can be used as a surrogate measure of preload.*
- Inotropes increase CO, thereby increasing MAP and maintaining perfusion to vital organs and tissues. Inotropes increase CO by increasing both SV and HR.
- In the failing heart, SV can only increase to a certain level before the cardiac muscle fibres become overstretched and CO will start to drop. (**Starling's law**).

1. DOBUTAMINE

- Dobutamine is **predominantly a β_1 agonist** and therefore increases cardiac contractility and heart rate.
- It also acts at **β_2 receptors** causing vasodilation and decreasing afterload.
- Because of this vasodilation, and to ensure adequate MAP is achieved, it may be necessary to administer dobutamine in combination with a vasopressor (eg, noradrenaline).
- **The main side effects** of dobutamine are increased heart rate, arrhythmias and raised myocardial oxygen demand. *These can cause myocardial ischaemia.*
- **Precautions**
 - *Avoid with systolic blood pressure < 100 mmHg and signs of shock.*
 - *May cause tachyarrhythmias, fluctuations in blood pressure, headache, and nausea.*
 - *Elderly patients may have a significantly decreased response.*
 - **DO NOT MIX WITH SODIUM BICARBONATE.**

2. ISOPRENALINE

- Isoprenaline has a similar profile to dobutamine but tends to cause more tachycardia.
- It is sometimes used for bradycardic patients requiring inotropic support.

3. NORADRENALINE

- Because noradrenaline **acts primarily via α_1 receptors**, it is usually used as a **vasopressor** (increasing SVR to maintain MAP) rather than an inotrope.
- It is often used with other inotropes, such as dobutamine, to maintain adequate perfusion, as discussed above.

4. ADRENALINE

- Adrenaline has activity at all adrenergic receptors (**predominantly acting as a β -agonist in low doses and an α -agonist at higher doses**); other more specific inotropes are often preferred over adrenaline.
- Adrenaline is used mainly during resuscitation after cardiac arrest (in this case it is given as a bolus).
- **It is not recommended for use in cardiogenic shock** because of metabolic side effects, including hyperlactataemia and hyperglycaemia.

5. DOPAMINE

- It is a complicated inotrope because it has dose-dependent pharmacological effects:
 - **Low-dose Dopamine (2–5 $\mu\text{g/kg/min}$):** Mainly Dopaminergic effects,
 - **Medium doses (5–10 $\mu\text{g/kg/min}$):** β_1 inotropic effects predominate
 - **High doses (10–20 $\mu\text{g/kg/min}$):** α_1 vasoconstriction predominates.

MECHANISM OF ACTION

- *Endogenous catecholamine, acting on both Dopaminergic and Adrenergic neurons:*
 - *Low dose stimulates mainly dopaminergic receptors, producing renal and mesenteric vasodilation; 1-5 mcg/kg/min IV: May increase urine output and renal blood flow.*
 - *Medium dose stimulates both beta1-adrenergic and dopaminergic receptors,*

producing cardiac stimulation and renal vasodilation; 5-10 mcg/kg/min IV: May increase renal blood flow, cardiac output, heart rate, and cardiac contractility

- **Large dose** stimulates alpha-adrenergic receptors; **10-20 mcg/kg/min IV**: May increase blood pressure and stimulate vasoconstriction; may not have a beneficial effect in blood pressure; may increase risk of tachyarrhythmias

6. PHOSPHODIESTERASE-3 INHIBITORS (MILRINONE)

- Phosphodiesterase-3 (PDE3) is an enzyme found in cardiac and smooth muscle cells.
- Inhibition of PDE3 increases intracellular calcium causing **vasodilation and increased myocardial contractility**.
- The mechanism of action is independent of adrenergic receptors and therefore PDE3 inhibitors are particularly useful if these receptors have become down-regulated (eg, in patients with chronic heart failure).
- **Milrinone** is the most commonly used PDE3 inhibitor. It has a relatively long half-life (**two hours**) and can accumulate in patients with renal failure.

7. LEVOSIMENDAN (unlicensed)

- Levosimendan is a novel inotrope that sensitises troponin-C to calcium, thereby increasing the force of contraction.
- It also acts on potassium channels in smooth muscle to cause vasodilation.
- Levosimendan increases CO without increasing myocardial oxygen consumption.
- Levosimendan is administered as a continuous infusion (with or without an initial bolus dose) over 24 hours.
- It has a half-life of about one hour, but active metabolites mean that the inotropic effect can continue for up to five days after the infusion has finished.

Agent	Receptor Agonist Activity*				Initial Dose	Onset
	α	$\beta 1$	$\beta 2$	DA		
Phenylephrine	++++	—	—	—	10 mcg/min	2 minutes
Norepinephrine	++++	+++	—	—	2 mcg/min	1-2 minutes
Epinephrine	+++	++++	+++	—	1 mcg/min	1 minute
Dopamine	++	++++	++	++++	5 mcg/kg per min	5 minutes
Dobutamine	+	++++	++	—	1 mcg/kg per min	1-2 minutes
Isoprenaline	—	++++	++++	—	5 mcg/min	1-5 minutes

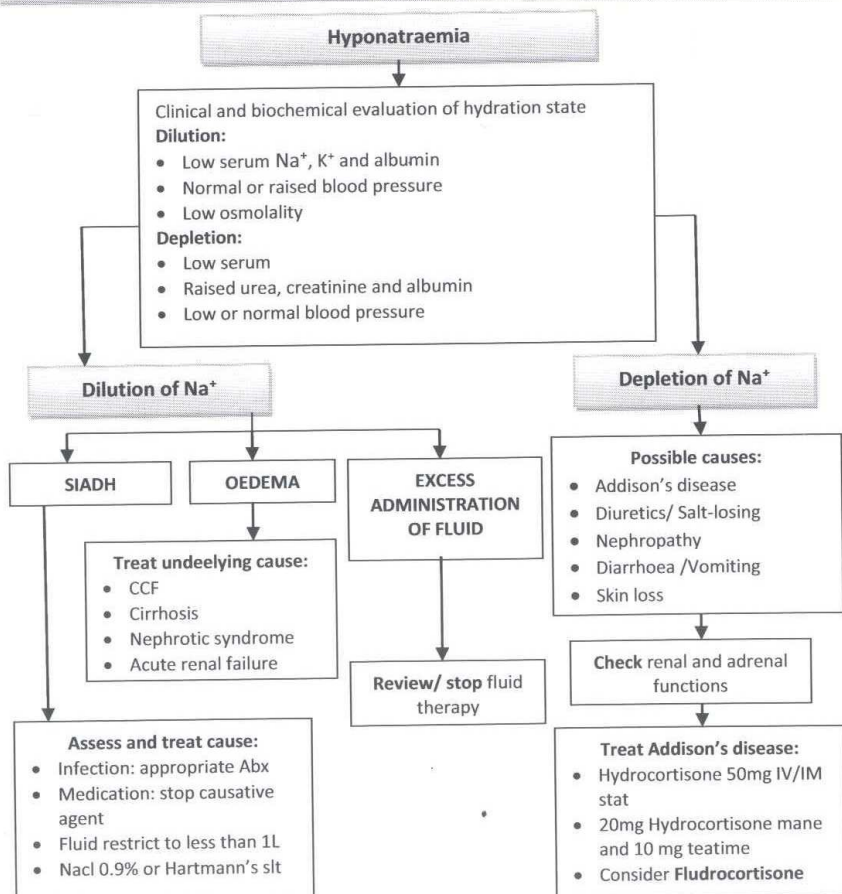
* Receptor activity may be dose dependent

CHAPTER 4. ELECTROLYTES IMBALANCES

I. HYPONATRAEMIA

Definition	Serum [Na ⁺] (mmol/l)
Hyponatraemia	< 135
Normal	135 - 145
Mild Hyponatraemia	146 - 149
Moderate Hyponatraemia	150 - 169
Severe Hyponatraemia	≥ 170

MANAGEMENT OF HYPONATRAEMIA



Maximum rate of change of serum Na concentration is 12mmol/l in 24hrs

SIADH

- Excess ADH results in inappropriate water retention by the kidneys leading to hyponatraemia.
- The diagnosis requires:
 - **Hyponatraemia + Low Serum Osmolality:** Less than 275mosm/Kg
 - **High urine osmolality/Concentrated urine:** Sodium >20 mmol/L and osmolality >300 mosm/kg
 - Absence of hypovolaemia, oedema, or diuretics.
- **CAUSES OF SIADH**
 - **CNS:** Meningitis, Encephalitis, Abscess, Stoke, Subarachnoid Haemorrhage, Subdural Haemorrhage, Head Injury.
 - **Malignancy:** Small Cell Lung Cancer, Pancreatic Cancer, Prostate Cancer, Lymphoma.
 - **Respiratory:** Pneumonia, Aspergillosis.
 - **Metabolic:** Porphyrria.
 - **Drugs:** Diuretics, Antidepressants, ACE Inhibitors, Antipsychotics, COX2 Inhibitors, PPI.
 - **Trauma.**
- **ED MANAGEMENT OF SIADH**
 - Treat the underlying cause
 - Cessation of offending drug.
 - Fluid restriction to less than 1l/day.
 - **Demeclocycline:** if persistent SIADH.
 - Urgent review by a renal consultant.

II. HYPERNATRAEMIA**CAUSES OF HYPERNATRAEMIA**

Excessive Water losses	Decreased Fluid Intake
<ul style="list-style-type: none"> ○ Renal: <ul style="list-style-type: none"> ▪ Central/Nephrogenic Diabetes insipidus ▪ Diuretics ▪ Tubulopathies ▪ Hyperglycaemia ○ Insensible: <ul style="list-style-type: none"> ▪ Fever or High ambient temperature ▪ Exercise ▪ Burns ▪ Respiratory illnesses ○ Gastrointestinal <ul style="list-style-type: none"> ▪ Gastroenteritis ▪ Osmotic Diarrhoea ▪ Colostomy/Ileostomy ▪ Malabsorption/ Vomiting 	<ul style="list-style-type: none"> ○ Neurologic impairment ○ Hypothalamic disorder ○ Restricted access to fluid ○ Fluid restriction ○ Ineffective breastfeeding
	<ul style="list-style-type: none"> ● Excess Sodium administration <ul style="list-style-type: none"> ○ Hypertonic NaCl ○ Sodium hydrochloride ○ Normal saline, blood products ○ Na ingestion

CLINICAL SIGNS/SYMPTOMS

- Severe symptoms mainly develop when the serum $[Na^+] > 160\text{mmol/L}$.
- More severe with acute hypernatraemia, Chronic hypernatraemia (present > 5 days) is often well tolerated because of cerebral compensation.
- Shock occurs late because intravascular volume is relatively preserved.
- Look for signs of intracellular dehydration and neurological dysfunction:
 - Lethargy, Irritability
 - Skin feels "doughy"
 - Ataxia, tremor
 - Hyperreflexia, Seizures, reduced GCS

INVESTIGATIONS

- Glucose, U&E, Plasma and Urine osmolality, Urinary Sodium concentration, TFT
- If seizures / neurological signs:
 - Recheck sodium urgently,
 - Consider neuroimaging,
 - Seek senior advice.

ED MANAGEMENT OF HYPERNATRAEMIA

- *Over rapid reduction of the sodium in hypernatraemia can cause cerebral oedema, convulsions and permanent brain injury.*
- *Over rapid correction of sodium in hyponatraemia can cause Central Pontine Myelinolysis with irreversible symptoms: dysarthria, Dysphagia, Tremor, Paraparesis or Quadriparesis, lethargy, confusion, Disorientation, Coma or Death.*
 - Close monitoring is critical. Resuscitation: If "shocked", resuscitate with boluses 20ml/kg of 0.9% saline as required.
- **Initial management and monitoring**
 - Fluid management should then be based on the initial serum sodium.
 - **Rate to lower sodium:** Aim to lower the serum sodium slowly at a rate of no more than 12mmol/L in 24 hours, (0.5mmol/L/hour).

III. HYPERCALCAEMIA

CONTEXT

- The adult body contains approximately 1kg of Ca^{2+} ions (Ca^{2+}) (25,000mmol) with over 99% bound in the skeleton. The 70 kg male contains approximately 22.5mmol of Ca^{2+} in extracellular fluid, of this only 9% is in plasma.
- Measurement**
 - The measurement of ionised Ca^{2+} in plasma is difficult and not routine practice.
- Formula**
 - Total plasma Ca^{2+} is corrected for protein binding by adding or subtracting 0.02mmol/l for every gram of albumin measured concurrently above or below 40g/l.
 - Corrected calcium (mmol/L) = $[\text{Ca}] \pm 0.02 (40 - \text{albumin})$**
 - Each 1 g/L decrease of albumin, will raise 0.02 mmol/L in serum Ca.
 - When there is hypoalbuminemia (a lower than normal albumin), the corrected calcium level is higher than the total calcium.
The formula is: $[\text{Ca}] + 0.02 (40 - \text{albumin})$
 - Thus, when there is a higher than normal albumin, the corrected calcium level is lower than the total calcium. The formula is: $[\text{Ca}] - 0.02 (40 - \text{albumin})$
 - Serum calcium level*
 - Normal: 2.2-2.6mmol/l
 - Hypercalcaemia: >2.6mmol/l
 - Hypocalcaemia: <2.2mmol/l
 - A patient's calcium is 2.55mmol/l and albumin is 35g/l. What is the patient's corrected calcium level?
 - Corrected calcium is: $2.55 + 0.02 (40 - 35) = 2.65\text{mmol/l}$
 - This indicates that hypercalcaemia is present.

Causes of Hypercalcaemia

- Nature:** Bones, Stones, Groans and Psychic Moans
- More than 90% from Malignancy and Primary Hyperparathyroidism

Malignancy	Endocrine	Drugs
<ul style="list-style-type: none"> Breast, Lung, Thyroid, Kidney, Prostate cancers Myeloma, Leukaemia Lymphoma: Hodgkin's and non-Hodgkin's 	<ul style="list-style-type: none"> Primary and Tertiary Hyperparathyroidism Addison's Phaeochromocytoma Hyperthyroidism 	<ul style="list-style-type: none"> Thiazides diuretics Lithium Theophylline toxicity Hypervitaminose A Hypervitaminose D
Other Causes		Fictitious
<ul style="list-style-type: none"> Rhabdomyolysis Respiratory: Sarcoidosis, TB Dehydration Immobilisation Milk-Alkali syndrome Familial hypocalciuric hypercalcaemia 		<ul style="list-style-type: none"> Not corrected level for albumin Prolonged cuff time Pagets (non-malignant increased bone turnover)

- **Clinical symptoms**
 - **Stones** (Renal colic and hypercalcaemic stones)
 - **Bones** (Increased osteolysis and fractures)
 - **Psychic Moans** (Depression, confusion, hallucinations and coma)
 - **Abdominal Groans** (Anorexia, Nausea & Vomiting, constipation, PUD, Pancreatitis)
 - **Other**
 - Muscle weakness, malaise, hyporeflexia
 - Confusion, apathy, decreased memory
 - Nephrogenic diabetes insipidus (Polyuria and polydipsia)

- **COMPLICATION OF HYPERCALCAEMIA**

- **Cardiac Arrhythmia**
- **ECG changes**
 - QT shortening
 - Prolonged PR
 - Widened QRS
 - Notched QRS with increased voltage
 - AV block
- **INVESTIGATIONS**
 - **Blood:** Calcium adjusted for albumin, Phosphate, PTH, U&E
 - **ECG**
- **High Ca and High PTH**= Primary and Tertiary hyperparathyroidism
- **High Ca and Low PTH**= Malignancy and other rarer causes

MANAGEMENT OF HYPERCALCAEMIA IN THE ED

- **Rehydration**
 - **IV Normal saline 0.9%** 4-6 liters in 24hrs
 - Consider **Haemodialysis** if severe Renal failure
 - *Must monitor or replace K and Mg as these will be lost in the urine along with the calcium*
 - **Do not give THIAZIDES**, they will worsen condition
 - *Treatment with FRUSEMIDE is controversial as it promotes Calcium bone reuptake*
- **After rehydration:**
 - **IV Biphosphonates:**
 - Zoledronic acid 4mg over 15min or
 - Pamidronate 30-90mg at 20mg/hr or
 - Ibandronic acid 2-4mg
- **Second line treatment**
 - **Prednisolone 40mg** daily (indicated in Lymphoma, other granulomatous diseases or 25-OHD poisoning)
 - **Calcitonin 4-8IU SC/IM BD** (if poor response to Biphosphonates)
 - **Calcimetics 30mg po BD** (indicated for primary hyperthyroidism, Parathyroid carcinoma or renal failure)
 - **Parathyroidectomy:** if poor response to other measures

IV. HYPOCALCAEMIA

• CAUSES

- **Low PTH**
 - Hypoparathyroidism
 - Hypomagnesaemia
 - Hungry bone syndrome
- **High PTH**
 - Vitamin D Deficiency: Dietary malabsorption or lack UVL
 - Renal failure: 1,25 OH D₃ synthesis impaired renal osteodystrophy; retention PO₄²⁻
- **Other causes**
 - Anticonvulsants
 - Massive transfusion
 - Osteomalacia/Rickets
 - Acute rhabdomyolysis
 - Acute pancreatitis
 - Parathyroid removal
 - Malabsorption e.g. coeliac disease
 - Di George syndrome
 - Pseudohypoparathyroidism: End organ resistance to PTH
 - Pseudo-Pseudohypoparathyroidism: Phenotypic above but normal Ca²⁺ metabolism.

• Features:

- Neuromuscular excitability
- Carpopedal spasm
- Tetany
- Chvostek's sign
- Trousseau's sign
- Seizures

• ECG CHANGES

- Hypocalcaemia causes QTc prolongation primarily by prolonging the ST segment.
- The T wave is typically left unchanged.
- Dysrhythmias are uncommon, although atrial fibrillation has been reported.
- Torsades de pointes may occur, but is much less common than with hypokalaemia or hypomagnesaemia.

• TREATMENT

- **Correction where acute plasma calcium is indicated:**
 - Promptly correct symptomatic or severe hypocalcaemia with cardiac arrhythmias or tetany with parenteral administration of calcium
 - **Start oral calcium and vitamin D treatment early**
 - Patients with cardiac arrhythmias or patients on **digoxin therapy** need continuous ECG monitoring during calcium replacement because *calcium potentiates digitalis toxicity*. Chronically, the treatment is aimed at the cause and may involve **alpha hydroxylated D₃** administration.
- Tetany may be caused by hypocalcaemia, alkalosis, hyperventilation, excess vomiting and primary hyperaldosteronism i.e. hyperchloraemic alkalosis.

CHAPTER 5. ENDOCRINE EMERGENCIES

I. ADRENAL INSUFFICIENCY

OVERVIEW

- Types:
 - Primary, secondary and tertiary + acute/chronic
- **Primary = Addison's**
 - Destruction of > 90% of adrenal glands
 - Rare
 - Causes: autoimmune destruction, haemorrhage, tumour (breast and melanoma), infection (TB, HIV, Meningococcaemia, Purpura Fulminans) or inflammatory process
 - Loss of mineralocorticoid and glucocorticoid activity
- **Secondary**
 - Insufficient production of ACTH
 - Rare
 - Mineralocorticoid function intact
 - Causes: destruction or dysfunction of the pituitary
- **Tertiary/Iatrogenic/Relative**
 - Suppression of HPA axis over time
 - Most common
 - Cause: administration of exogenous glucocorticoids
 - Mechanism: chronic ACTH suppression → adrenal atrophy

1. ADDISON'S DISEASE

PHYSIOLOGY OF THE ADRENAL GLAND

- The adrenal gland is divided into the cortex and medulla.
- The cortex is divided into three zones:
 - **Zona glomerulosa:** this is under regulation by the renin-angiotensin system and produces and secretes mineralocorticoids, mainly aldosterone.
 - **Zona fasciculata:** this is under regulation by the hypothalamic-pituitary axis. It produces and secretes glucocorticoids, mainly cortisol, in response to adrenocorticotrophic hormone (ACTH) from the anterior pituitary.
 - **Zona reticularis:** this produces and secretes androgens.
- The adrenal medulla receives input from the sympathetic nervous system and secretes catecholamines, mainly adrenaline and noradrenaline.

CAUSES OF ADRENAL INSUFFICIENCY

- **In primary adrenocortical insufficiency:** Most cases of Addison's disease are caused by the gradual destruction of the adrenal cortex by the body's own immune system. *Glucocorticoid and Mineralocorticoid functions are lost.*
- **In secondary adrenocortical insufficiency:** mineralocorticoid function is preserved because the insufficiency is due to disease or suppression of the hypothalamic-pituitary axis.

CAUSES OF ADRENAL INSUFFICIENCY

Primary causes	Secondary causes
<ul style="list-style-type: none"> • Idiopathic/Autoimmune • Infective: TB, AIDS, Fungal infection. • Haemorrhage: anticoagulant therapy, Waterhouse–Friderichsen syndrome (haemorrhage into the adrenal gland secondary to fulminant meningococcal septicaemia). • Infiltration: carcinoma, lymphoma, sarcoidosis, amyloidosis. • Drugs: ketoconazole, Etomidate 	<ul style="list-style-type: none"> • Abrupt withdrawal of long term steroids • Trauma to infundibular stalk • Necrosis (Sheehan's syndrome) • Neoplasms and granulomatous disease of pituitary • Radiation to pituitary

INVESTIGATION OF ADRENOCORTICAL INSUFFICIENCY

- U&E, Serum Cortisol & Plasma ACTH
- Infective Screen
- ECG
- **Adrenocortical deficiency results in:**
 - Hyponatraemia.
 - Hyperkalaemia.
 - Hypoglycaemia.
 - Elevated urea and creatinine.
 - Metabolic acidosis.
 - Serum cortisol and plasma ACTH levels should be sent, but should not delay treatment with hydrocortisone.
- Interpretation of the cortisol and ACTH results:
 - Low serum cortisol (<200nmol/L): indicates adrenal insufficiency.
 - A raised ACTH in this context suggests primary adrenal insufficiency and a low ACTH suggests secondary.
 - High serum cortisol (>550nmol/L): excludes adrenal insufficiency.
 - Intermediate serum cortisol (200–550 nmol/L): requires further investigation with a Synacthen (tetracosactrin) test.

MANAGEMENT OF ACUTE ADRENOCORTICAL INSUFFICIENCY

- Treatment of a suspected adrenal crisis should not be delayed pending the results of cortisol and ACTH.
- ABCD Approach
- Hydrocortisone 100 mg IV should be given as soon as an adrenal crisis is suspected.
- Fludrocortisone is only required in primary adrenocortical insufficiency and is not commonly given in the ED.
- Fluid resuscitation should be directed by cardiovascular status.
- Patients should be monitored for hypoglycaemia and treated with 10% glucose IV if it develops.
- Any underlying infection should be treated with appropriate antibiotics.

II. PHAEOCHROMOCYTOMA

• INTRODUCTION

- Pheochromocytomas are rare neuroendocrine tumours that arise from either adrenal medulla or extra adrenal chromaffin tissue.
- They can produce a variety of nonspecific symptoms, which include headaches, sweating, anxiety and palpitations.
- Pheochromocytoma is associated with Von Hippel Landau disease, Multiple Endocrine Neoplasia (MEN) type 2 syndromes and Neurofibromatosis type 1.
- 80% are unilateral and solitary, 10% are bilateral and 10% are extra-adrenal.
- Approximately 90% are benign and 10% are malignant.
- Common signs include hypertension and tachycardia.
- Surgery, especially adrenal laparoscopy, is the most common treatment for small pheochromocytomas.

• PATHOPHYSIOLOGY

- The manifestations of pheochromocytoma are due mostly to the increased abnormal secretion of catecholamines, principally epinephrine, but also norepinephrine and dopamine.
- The relative amounts of catecholamines secreted can differ between tumours and this determines the clinical picture. The catecholamines can also be released episodically.
- The effects of epinephrine and norepinephrine are caused by agonist activity at alpha and beta adrenoceptors and are detailed above (see vasoactive Drugs).

• CLINICAL ASSESSMENT

- The presenting features of pheochromocytoma are very wide and varied.
- For this reason it is referred to as the "great mimic".
- Hypertension is a common presenting feature with SBP>220 mmHg or DBP<120 mmHg being generally accepted limiting values.
- Hypertension is frequently associated with profound tachycardia, pallor and a feeling of anxiety or impending doom. These symptoms are often paroxysmal and can occur many times a month or just once with a single fatal presentation.
- The diagnosis should be considered in any patient presenting with acute hypertension or with a hypertensive crisis but be aware that hypertension can be episodic or absent and consider the diagnosis if there is a syndrome of appropriate clinical features compatible with the diagnosis.
- Precipitants can include abdominal compression, anaesthesia, opiates, dopamine antagonists, cold medications, radiographic contrast media, catecholamine reuptake inhibitors and childbirth.

• DIFFERENTIAL DIAGNOSIS OF PHAEOCHROMOCYTOMA

Endocrine	Cardiovascular	Neurological	Miscellaneous
<ul style="list-style-type: none"> • Hyperthyroidism • Carcinoid • Hypoglycaemia • Medullary thyroid carcinoma • Mastocytosis • Menopausal syndrome 	<ul style="list-style-type: none"> • Heart failure • Arrhythmias • IHD • Baroreflex failure • Renovascular hypertension 	<ul style="list-style-type: none"> • Migraine • Stroke • Diencephalic epilepsy • Meningioma • Postural orthostatic tachycardia syndrome 	<ul style="list-style-type: none"> • Essential hypertension • Alcohol withdrawal • Pre-eclampsia • Porphyria • Panic Disorder or Anxiety • Factitious Disorders • Drug treatment • Illegal Drug Use

INVESTIGATION STRATEGIES

- ECG, Capillary Blood Glucose and FBC.
- CT scan of the abdomen & MRI: Sensitivity 93-100%
- Specific investigation for phaeochromocytoma is not usually instigated in the ED; appropriate subsequent tests include assay of plasma and urine metanephrines, catecholamines and urine vanillylmandelic acid (VMA).
- The most sensitive test is Plasma Metanephrine Assay (99% sensitivity with a specificity of 89%).

MANAGEMENT OF PHEOCHROMOCYTOMA

- Definitive treatment is by **surgical resection of the tumour**, normally using a laparoscopic approach.
- Prior to surgery the acute crisis is treated medically to control the effects of excess catecholamines.
- This is normally achieved by alpha adrenoceptor blockade.
- **Phenoxybenzamine** is advocated as it blocks adrenoceptors irreversibly and therefore its effect cannot be overcome by increasing catecholamine concentrations.
- **Phentolamine and Doxazosin** are alternative alpha antagonists.
- **Phenoxybenzamine IV 10-40 mg over one hour.** It acts within one hour and its effects last for up to four days. It can be given orally in a dose of 10-60 mg/day in divided doses.
- Side effects include hypotension, dizziness, sedation, dry mouth, paralytic ileus and impotence.
- **Phentolamine 5-10 mg:** used in the diagnosis and perioperative management of phaeochromocytoma. It causes vasodilatation, but also has positive inotropic and chronotropic effects. It exerts its effect predominantly by competitive alpha adrenoceptor blockade.
- **Side effects** include orthostatic hypotension, dizziness, abdominal discomfort and diarrhoea. Cardiovascular collapse has occurred following treatment of phaeochromocytoma.
- Beta adrenoceptor blockade can be instituted to control tachycardia, but this should only be done after adequate alpha blockade, otherwise unopposed alpha activity can lead to worsening hypertension.
- Treat arrhythmias if indicated.
- IV fluid if fluid depleted.

III. THYROID EMERGENCIES

1. THYROID STORM

• DESCRIPTION

- **Malignant or critical thyrotoxicosis, thyroid storm**, is a life-threatening medical emergency in which excessive concentrations of thyroid hormone produce organ dysfunction. It is an uncommon manifestation of hyperthyroidism, occurring in less than 10% of patients hospitalized for thyrotoxicosis. However, it may be the presenting symptom of the condition and, if untreated, is associated with 80% to 90% mortality.
- Even with treatment, mortality from thyroid storm exceeds 20%.
- Recognition and immediate management is important in preventing the high morbidity and mortality associated with this disease.
- A spectrum of thyroid dysfunction exists. Hyperthyroidism, or thyrotoxicosis, refers to disorders that result from overproduction and release of hormone from the thyroid gland. Thyrotoxicosis refers to any cause of excessive thyroid hormone concentration, whereas malignant thyrotoxicosis, or thyroid storm, represents an extreme manifestation of thyrotoxicosis with resultant end-organ dysfunction.
- Incidence Thyroid storm can occur in both men and women of any age. However, it is more common in teenaged or young adult women. Although a history of hyperthyroidism is common, thyroid storm may be the initial manifestation in a significant number of patients.
- Thyroid storm can be precipitated by a variety of factors, including severe infection, diabetic ketoacidosis, surgery, trauma, and pulmonary thromboembolism.
- Direct trauma or surgical manipulation of the thyroid gland can also precipitate thyroid storm. Iodine, either from excessive ingestion, intravenous administration, or radiotherapy, has been reported to precipitate thyroid storm.
- It has also been described following discontinuation of antithyroid medications.
- Of interest, salicylates have been implicated in triggering thyroid storm by increasing the concentration of circulating free thyroid hormones to critical levels.

• CLINICAL MANIFESTATION AND DIAGNOSIS

- The clinical manifestations of thyroid storm are consistent with marked hypermetabolism resulting in multiorgan system dysfunction.
- The differential diagnosis of thyroid storm includes sepsis, central nervous system infection, anticholinergic or adrenergic intoxication, other endocrine dysfunction, and acute psychiatric illness.
- Symptoms include thermoregulatory dysfunction (high fever, warm moist skin, diaphoresis), neurologic manifestations (mental status changes, seizure, coma, psychosis, hyperreflexia, lid lag), cardiovascular dysregulation (atrial fibrillation, tachycardia, hypertension, congestive heart failure), respiratory distress (dyspnea, tachypnea), and gastrointestinal dysfunction (diarrhea, abdominal pain, nausea, vomiting). The diagnosis of thyroid storm relies heavily on clinical suspicion. It is strongly suggested by the constellation of these symptoms and is confirmed by means of thyroid function tests (TFT).
- However, treatment should not be delayed for verification by laboratory tests. Thyroid stimulating hormone (TSH) levels are virtually undetectable (<0.01 micro international units [mIU]/L) with a concomitant elevation of free T4 and T3.

- Because of increased conversion of T₄ to T₃, the elevation of T₃ is typically more dramatic. For this reason it is essential to measure both T₃ and free T₄ levels when thyroid storm is suspected. There are no differences in the results of TFT in patients with thyroid storm when compared with patients who have symptomatic hyperthyroidism, and levels of thyroid hormone cannot predict which patients will undergo decompensation from thyrotoxicosis to thyroid storm.
- The distinction is made clinically by documentation of acute organ dysfunction. Other laboratory abnormalities commonly seen are hypercalcemia from osteoclast-mediated bone resorption, elevated alkaline phosphatase caused by activated bone remodeling, and hyperglycemia secondary to enhanced glycogenolysis and increased circulation of catecholamines. Adrenal insufficiency, especially among patients with Graves disease, is common and should be evaluated prior to the initiation of treatment.

● TREATMENT

- The treatment of thyroid storm involves 3 critical fundamentals:
 - **First**, supportive care should be provided to minimize the secondary effects of organ failure. This should include respiratory and hemodynamic support and treatment of hyperthermia.
 - **Second**, identification and treatment of the precipitating event is warranted to prevent further progression of disease.
 - **Third**, and most critical, the release and effects of circulating thyroid hormone must be blocked. Inhibition of the peripheral conversion of T₄ to T₃ helps attenuate the effects of thyroid hormone.
- **Propylthiouracil (PTU)** blocks peripheral conversion of T₄ to T₃ and can be given as a 600- to 1000-mg loading dose, followed by 1200 mg/day divided into doses given every 4 to 6 hours.
- **Methimazole** can be used as an alternate agent but does not block peripheral T₄ conversion. Both medications can be administered rectally if necessary.
- Peripheral thyroid hormone action as well as tachycardia and hypertension can be minimized by beta-blockers; typically **Propranolol** administered intravenously initially in 1-mg increments every 10 to 15 minutes until symptoms are controlled or **Esmolol** administered as a loading dose of 250-500 mcg/kg followed by an infusion of 50-100 mcg/kg/minute.
- Thyroid hormone release can be reduced by the administration of **lithium, iodinated contrast, and corticosteroids**.
- **Hydrocortisone 100 mg** given intravenously every 8 hours has been shown to improve outcomes in patients. Steroid therapy is also beneficial, given the common association with adrenal insufficiency. Iodine acts by inhibiting hormone release but should not be given until 1 hour after PTU administration. In refractory cases, plasmapheresis, plasma exchange, and peritoneal hemodialysis can be used to remove circulating thyroid hormone.
- With appropriate treatment, clinic and biochemical improvement are typically seen within 24 hours.
- Full recovery usually occurs within a week of therapy. Thyroid storm poses diagnostic and therapeutic challenges.
- Treatment is aimed at halting the thyrotoxic process at all levels. Prompt recognition and treatment is essential for successful management and is paramount to decreasing the high mortality associated with this disease.

2. MYXEDEMA COMA**• DESCRIPTION**

- Myxedema coma is an uncommon presentation of severe hypothyroidism that is potentially fatal. Published mortality rates exceed 60%, and even with early detection and appropriate treatment, death occurs in up to 30% of individuals.
- The term myxedema coma is a misnomer, as myxedema and coma are neither diagnostic criteria nor common presenting findings. A more proper description would be critical hypothyroidism.
- Because of its lethal nature and nonspecific features, the actual prevalence of myxedema coma is unknown. However, this syndrome is extensively cited in the literature and is not uncommon in clinical practice.

• INCIDENCE

- Myxedema coma, or critical hypothyroidism, occurs most often in patients with long-standing, preexisting hypothyroidism. Hypothyroidism is 4 times more common in women than in men, and 80% of cases of myxedema coma occur in females.
- It occurs almost exclusively in persons 60 years or older. There are approximately 300 cases of myxedema coma reported in the literature.
- Most cases occur during the winter, when thermoregulatory stressors are high.
- It can develop from all causes of hypothyroidism, including autoimmune thyroiditis, secondary hypothyroidism, and drug-induced hypothyroidism (eg, caused by lithium or amiodarone).

• CLINICAL MANIFESTATION AND DIAGNOSIS

- Myxedema coma can be precipitated by several factors. Infections, especially pneumonia, are perhaps the most common precipitating factor.
- Even occult bacterial infections have been implicated and, as such, infections should be thoroughly evaluated for as a potential etiologic factor.
- Cardiac events (myocardial infarction, congestive heart failure), cerebral infarction, trauma, hemorrhage, hypothermia, hypoglycemia, and respiratory depression secondary to anesthetics or sedatives have also been implicated.
- Clinical findings in myxedema coma are similar to those encountered with hypothyroidism, but they are typically seen in greater magnitude. In short, it is a state of profound decreased metabolic activity.
- Cardinal features include impaired thermoregulation (hypothermia), hypotension, bradycardia, and mental status depression.
- Mental status depression is a common clinical feature and may progress to stupor, obtundation, or frank coma. The hypometabolic state and mental status depression may result in centrally mediated hypoventilation and hypercapnic respiratory failure.
- Concomitant endocrinopathies are commonly encountered, most notably adrenal insufficiency, which may contribute to the electrolyte, thermoregulatory, and cardiovascular derangements commonly seen. Hyponatremia resulting from an increased release of antidiuretic hormone and hypoglycemia caused by decreased gluconeogenesis, infection, or adrenal insufficiency are common features.
- Myxedema is characterized by generalized skin and soft tissue swelling, periorbital edema, ptosis, macroglossia, and the presence of cool, dry skin.
- Despite the name of the condition, clinically significant myxedema is infrequently identified and is not a diagnostic criteria.

- Unlike thyroid storm, most patients with myxedema coma have a prior diagnosis of hypothyroidism. Although it is necessary to confirm the diagnosis, thyroid function testing can be confusing. The diagnosis is suspected clinically and confirmed with TFT. Treatment should not be delayed for laboratory confirmation.
- Hypothyroidism is diagnosed in individuals with elevated TSH levels and low levels of free T4 and T3. In myxedema coma, T3 and T4 levels may be profoundly diminished or even undetectable.
- The degree of TFT abnormalities does not distinguish hypothyroidism from myxedema coma. Rather, the distinction is based on clinical findings. Abnormal TFT can be seen in other acute illnesses and does not necessarily reflect myxedema coma or even hypothyroidism. It is important for the clinician to be able to differentiate hypothyroidism from euthyroid sick syndrome, in which patients have a reduction in both TSH and thyroid hormone levels.
- Given the common association with adrenal insufficiency, a cosyntropin stimulation test should be considered, especially in those with hemodynamic instability.

• TREATMENT

- The treatment of myxedema coma involves rapid replacement of thyroid hormone, treatment of the precipitating cause, and general supportive measures. A stated, despite a prompt diagnosis and initiation of treatment, mortality from myxedema coma can still exceed 30%. Thyroid hormone replacement should be given intravenously to ensure rapid restoration of bioactive thyroxine levels and resolution of symptoms. Both high-dose and low-dose strategies have been used. However, neither has been shown to be superior.
- High-dose intravenous thyroxine is given as a bolus of 300-500 mcg, followed by 50-100 mcg daily depending on the patient's age, weight, and risk of complications.
- This method provides a more rapid recovery of symptoms but carries the potential for unwanted cardiac events resulting from the rapid replacement of thyroxine.
- In the low-dose method, thyroxine 25 mcg is given daily for 1 week followed by a gradually increased dose until the patient is able to resume normal thyroxine orally.
- Alternatively, 5 mcg of triiodothyronine can be given twice daily during the loading period. Intravenous triiodothyronine can be used as well and may provide a more rapid resolution of symptoms and improved mental status, although high levels of triiodothyronine have been correlated with increased mortality.
- Triiodothyronine is given as an initial bolus dose of 10-20 mcg, followed by 10 mcg every 4 to 24 hours, with taper to 10 mcg every 6 hours.
- Regardless of the replacement method used, all patients should be continuously monitored for hypertension and cardiac ischemia, which portend the greatest risk of death among patients with myxedema coma.
- Treatment should also be directed at identifying and reversing the underlying cause. Supportive care should be provided while thyroid hormone levels are replaced. Ventilatory support, passive external rewarming, and correction of underlying electrolyte abnormalities are commonly required.
- Glucose and steroid replacement should also be considered until recovery.
- Given the strong association with infectious causes, antimicrobial therapy should be considered. Myxedema coma is a potentially fatal complication of a common disorder.
- Prompt recognition based on clinical features and institution of aggressive comprehensive treatment can reduce mortality.

IV. PITUITARY DISEASE

INTRODUCTION

- The pituitary is an endocrine gland located in the sella turcica in the skull base. Superior to it is the hypothalamus and the optic chiasm, laterally is the cavernous sinus through which run the III, IV, V, and VI cranial nerves.
- The pituitary is divided into anterior and posterior parts. The anterior pituitary produces and secretes hormones; it is regulated by hypothalamic hormones and negative feedback from target organs. The posterior pituitary is mainly a neuronal extension of the hypothalamus and secretes hormones made in the hypothalamus.
- Hormones secreted by the anterior and posterior pituitary

Anterior pituitary	Posterior pituitary
<ul style="list-style-type: none"> • Adrenocorticotrophic hormone (ACTH) • Growth hormone (GH) • Follicle-stimulating hormone (FSH) • Luteinizing hormone (LH) • Thyroid-stimulating hormone (TSH) • Prolactin (PRL) 	<ul style="list-style-type: none"> • Anti-diuretic hormone (ADH) • Oxytocin

1. PITUITARY APOPLEXY

- Pituitary apoplexy is caused by acute haemorrhage or infarction of the pituitary gland. An existing pituitary adenoma is usually present. The anterior pituitary gland has an unusual vascular supply being perfused by a portal venous system, making it an area prone to infarction.
- **PREDISPOSING FACTORS**
 - Head trauma.
 - Anticoagulation.
 - Pituitary radiotherapy.
 - Endocrine stimulation tests.
 - Drugs, e.g. oestrogens, bromocriptine.
- **Sheehan's syndrome:** during pregnancy the pituitary hypertrophies, however the blood supply from the low-pressure portal venous system remains unchanged. If major haemorrhage or hypotension occurs during the peripartum period the anterior pituitary may infarct. The posterior pituitary is usually spared due to its direct arterial blood supply.
- **CLINICAL FEATURES**
 - Severe headache.
 - Nausea, vomiting.
 - Photophobia
 - Loss of consciousness.
 - Meningism.
 - Visual field defect—bitemporal hemianopia.
- Cranial nerve palsies:
 - CN III (unilateral dilated pupil, ptosis, and a globe deviated inferiorly and laterally),
 - CN IV (inability to look down and in, resulting in vertical diplopia),
 - CN V (facial pain or sensory loss),
 - CN VI (unable to abduct eye, resulting in horizontal diplopia).

- Patients may have a history suggestive of pre-existing endocrine dysfunction (e.g. amenorrhoea, hypogonadism, decreased libido, obesity, lethargy, constipation, etc.).
- **INVESTIGATIONS**
 - CT or MRI head.
 - Blood should be taken to measure pituitary hormones (ACTH, TSH, FSH, LH, and prolactin) and the effects these hormones have on target organs (oestradiol—women, testosterone—men, T4, T3, cortisol).
 - Electrolytes and glucose should be monitored.
- **MANAGEMENT**
 - Hydrocortisone 100 mg intravenously 6-hourly.
 - Supportive therapy (ABCDE).
 - Urgent neurosurgical opinion.

2. CUSHING'S SYNDROME

- Cushing's syndrome is a debilitating endocrine disorder characterized by excessive cortisol levels in the blood which may be the result of a tumor of the pituitary gland, adrenal glands (located above the kidneys) or from tumors or cancer arising elsewhere in the body (ectopic ACTH producing tumors).
- Cushing's disease refers specifically to excessive ACTH secretion by a pituitary tumor (also called pituitary adenoma).
- The cause of Cushing's Syndrome is a pituitary adenoma in over 70% of adults and in approximately 60-70% of children and adolescents. Most pituitary ACTH-secreting adenomas are small in size (microadenomas).
- Overall, Cushing's Disease is relatively rare, affecting 10 to 15 of every million people each year, and most commonly affects adults aged 20 to 50 years. Women account for over 70% of cases.
- **SYMPTOMS AND SIGNS:**
 - Change in body habitus: weight gain in face (moon face), above the collar bone (supraclavicular) and on back of neck (buffalo hump)
 - Skin changes with easy bruising, purplish stretch marks (stria) and red cheeks (plethora)
 - Excess hair growth (hirsutism) on face, neck, chest, abdomen, and thighs
 - Generalized weakness and fatigue
 - Loss of muscle
 - Menstrual disorders in women (amenorrhoea)
 - Decreased fertility and/or sex drive (libido)
 - Hypertension
 - Diabetes mellitus
 - Depression with wide mood swings
- **DIAGNOSIS**
 - Comparison of old and recent photographs will often demonstrate the marked changes in facial appearance and body habitus of patients who develop Cushing's syndrome or Cushing's disease.

- However, the diagnosis of Cushing's disease is often long delayed and can be difficult to make. An endocrinologist should always supervise the evaluation for Cushing's disease.

- **Hormonal Diagnosis:**

- The first step in diagnosing Cushing's disease is to confirm the presence of excessive cortisol secretion (Cushing's syndrome). This diagnosis is most easily made by performing a low-dose dexamethasone suppression test or a 24-Hour urine collection to quantitate cortisol levels. The low-dose dexamethasone suppression test involves taking a small dose of dexamethasone (1mg) at 11 pm and having blood drawn for cortisol the following morning at 8 am.
- Once the diagnosis of Cushing's syndrome is established, the source of the excess cortisol needs to be determined: either from an adrenal gland tumor, an ectopic ACTH-producing tumor or a pituitary ACTH-producing adenoma. A high dose dexamethasone test, ACTH levels, metyrapone test, and/or sometimes a CRH test are used for this determination. In some individuals with depression, alcohol abuse, anorexia nervosa or high estrogen levels, cortisol levels may be chronically elevated.
- These patients with "pseudo-Cushing's" may be difficult to distinguish from those with true Cushing's disease.
- Additional hormonal tests are often needed to clarify the diagnosis in these individuals.

- **Petrosal Sinus Sampling:**

- Petrosal Sinus Sampling is an angiographic and endocrinological test used to distinguish between ectopic ACTH production or pituitary ACTH production (Cushing's disease).
- It is also used to help determine on which side of the pituitary gland an adenoma is located in patients with normal MRIs of the pituitary but with hormonal studies strongly suggesting Cushing's disease.
- Petrosal sinus sampling should never be performed before the diagnosis of Cushing's syndrome is established.

- **Imaging:**

- If laboratory tests suggest a pituitary adenoma as the cause of Cushing's, then a pituitary MRI is performed to confirm the diagnosis.
- In approximately 70% of cases an adenoma can be seen.
- A CT scan will detect only about 50% of adenomas.
- CT scans of the adrenal glands are very useful for determining the presence or absence of an adrenal tumor causing Cushing's syndrome.

- **TREATMENT**

- Cushing's syndrome does not require acute treatment in the ED.
- However, patients with Cushing's syndrome are more prone to fractures, infections, and poor wound healing, so may present with complications that require treatment.
- If the cause of Cushing's syndrome is exogenous steroids, these may be gradually tapered off and eventually stopped, if possible.
- Definitive treatment for Cushing's disease is selective removal of the pituitary adenoma.
- If the source cannot be located, bilateral adrenalectomy may be required.

V. DIABETES INSIPIDUS

- Diabetes insipidus (DI) is due to impaired water resorption by the kidney because of reduced secretion of ADH from the posterior pituitary (cranial DI) or impaired response of the kidney to ADH (nephrogenic DI).

• CAUSES OF DIABETES INSIPIDUS

Cranial DI	Nephrogenic DI
<ul style="list-style-type: none"> • Head injury • Hypophysectomy • Meningitis • Pituitary tumour • Metastases • Craniopharyngioma • Vascular lesion • Idiopathic (50%) 	<ul style="list-style-type: none"> • Low potassium • High calcium • Drugs (e.g. lithium) • Pyelonephritis • Hydronephrosis • Polycystic kidney disease • Inherited

• CLINICAL FEATURES

- Polyuria.
- Polydipsia.
- Dilute urine.
- Dehydration.

• INVESTIGATION

- Plasma osmolality—high.
- Urine osmolality—low.
- Serum sodium—high.
- Check serum potassium and calcium as potential causes.
- CT head if a cranial cause suspected.
- Measure pituitary function (TSH, ACTH, LH, FSH, and Prolactin).

• EMERGENCY TREATMENT

- Cranial DI—desmopressin 1mcg intranasally.
- Nephrogenic DI—treat the cause.
- Rehydrate—match input to fluid losses and aim to gradually reduce the serum sodium.

MAJOR TRAUMA & FRACTURES

6 QUESTIONS

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CHAPTER 1. BURNS

I. BURN DEPTHS AND EXTENT OF BURNED AREA

- In the treatment of burns the first thing to be described is the **depth of the burn** and the **proportion of the body being involved**.
- In this way, the severity can be clarified and the treatment designed.
- The classification of burn depth has throughout several years been under debate.
- Most often used terms are depths related to thickness or to degree.

A. BURN DEPTHS

DEPTHS	CLINICAL
Superficial epidermal burn	Involve the Epidermis Erythema, slightly swollen and painful, but not blistered. Should not be calculated in the extent of the burned surface area.
Superficial burn Dermal	Involve Epidermis and part of the Dermis . Skin is pale pink and painful, and there may be small blisters
Deep burn Dermal (Partial thickness)	Involve epidermis, the entire Dermis down to reticular dermis . Skin turns red and blotchy; dry or moist, swollen and blistered, and very painful or painless.
Full thickness burn	All three layers of skin (the epidermis, dermis and subcutis) are damaged; Skin is often burnt away and the tissue underneath may appear pale or blackened, while the remaining skin will be dry and white, brown or black with no blisters, and the texture of the skin may also be leathery or waxy

B. THE EXTENT OF THE BURN

- **Methods**
 - *Rules of 9's*
 - *Palm of patient = 1% TBSA burn*
 - *Lund-Browder Chart*

C. BURN SHOCK RESUSCITATION

- Burn shock resuscitation is defined as a controlled I.V. fluid administration securing vital organ function at the least physiological cost.
- The purpose of burn shock resuscitation is to counteract the hypovolemia seen during the first 24-48 hours after the trauma.
- A profound fluid shift in the body takes place even though a total body water can remain unchanged. However, evaporative water loss from the burned areas is massive.
- Burned patients requiring burn shock resuscitation should always be transferred to a burn centre.
- Different I.V. fluid formulas only serve as guidelines for starting up the resuscitation.
- Burn shock resuscitation is required if the % burned surface area exceeds:
 - *10% for children*
 - *15% for adults*
 - *10% for the elderly (more than 65 years of age)*

D. TRANSFERRAL CRITERIA TO A BURN CENTRE

- Patients requiring burn shock resuscitation.
- Burns > 10 % TBSA in an Adult
- Burns > 5 % TBSA in a Child
- Full thickness burns > 5% TBSA
- Burns of face, hands, feet, perineum, genitalia, and major joints
- Circumferential burns
- Chemical or electrical burns
- Burns in the presence of major trauma or significant co-morbidity
- Burns in the very young patient, or the elderly patient
- Burns in a pregnant patient
- Suspicion of Non-Accidental Injury

E. SMOKE INHALATION INJURY

- Common signs of **SIGNIFICANT SMOKE INHALATION INJURY** and the potential need for intubation include:
 - *Persistent cough, stridor, or wheezing*
 - *Hoarseness*
 - *Nares with inflammation or singed hair*
 - *Carbonaceous sputum or burnt matter in the mouth or nose*
 - *Blistering or edema of the oropharynx*
 - *Deep facial or circumferential neck burns*
 - *Depressed mental status, including evidence of drug or alcohol use*
 - *Respiratory distress*
 - *Hypoxia or hypercapnia*
 - *Elevated carbon monoxide and/or cyanide levels*

It's 4 am on a steady night shift in your regional Emergency Department when the bat phone rings...A 24-year-old male has been involved in a house fire. He has burns to his anterior chest, face, neck, and right arm. These are his vitals: HR 120 BP 100/62 RR 25 Sats 95% GCS 10. Are you ready to deal with a major burns patient? You'd better be, the ambulance bay doors are about to open.

Q1. How are you going to approach the preparation, assessment and management of this patient?

- The approach to the patient with a major burn is the same as the patient who has sustained any other type of major trauma, the ABCDE approach.
 - Airway maintenance with cervical spine protection
 - Breathing and ventilation
 - Circulation with hemorrhage control
 - Disability
 - Exposure and environmental control

Q2. What is a major burn?

- One definition suggests that those burns **requiring fluid resuscitation**, or with an **inhalational component** be considered major burns.
- Other definitions are similar to those injuries requiring Burns Centre Referral, see above.

Q3. What are the specific things to consider in the assessment of the major burns patient?

- When dealing with a patient with major burns, there are special considerations that need to be occurring during the ABCDE approach.

A – Airway

- Don't forget C-Spine immobilisation
- Burns are a major distracting injury and patients with burns are at risk of c-spine injuries e.g. jumping from burning building, explosions, and lightning strikes.
- Assess for evidence of airway burns e.g. singed facial hair, soot in the nose or mouth, stridor, voice change.
- Assess for evidence of neck burns / swelling that might impede airway
- Consider early intubation if evidence of airway compromise

B-Breathing

- All burns patients should have high flow oxygen 15L/min via non-rebreather mask
- Assess for the presence of constrictive chest wall burns. Assess for presence of toxic gas inhalation particularly carbon monoxide and cyanide toxicity

C-Circulation

- Place IV cannulae through unburnt skin where possible
- Assess for circumferential burns to limbs
- Shock due to burns is uncommon in the early phase and if present other causes should be sought e.g. Tension Pneumothorax, Abdominal Injury, Spinal injury etc.

D-Disability

- Remember hypoxia and toxic gas inhalation can result in altered mental status

E-Exposure

- Caution with risk of hypothermia especially in children
- Remove jewellery and burnt / wet clothes (see first aid below)
- The presence of circumferential burns to limbs or the chest may result in mechanical compromise leading to limb ischaemia or difficulty in ventilation. In these scenarios an **escharotomy** may be indicated, although rarely performed in the ED unless significant delays to definitive burns care is anticipated.
- Early discussion with regional burns unit is advised if an escharotomy appear indicated.

Q4. How do you assess a burn wound?

- Burns Wound Assessment is a two-part process consisting of:
 - Estimating Total Body Surface Area (TBSA) % of Burn
 - Estimating Depth of Burn

Q5. How should burn TBSA % be estimated?

- Estimating the area of a burn is difficult, especially in the early phase after injury.
- Erythema (epidermal) depth burns are NOT included in the estimation of burns area.
- Options for calculating burn area include:
 - **Palmar Surface**
 - The patient 's palmar surface, palm and fingers, is approximately 1% of their TBSA. This can be used to estimate the size of smaller burns or used to measure unburnt skin in large burns
 - **Rule of Nines**
 - Divides the body into 11 areas each of 9% TBSA, and the perineum ~1%.
 - Allows quick assessment in the Adult burns patient
 - **Paediatric Burns Area Assessment**
 - Children have a larger head TBSA % and a smaller leg TBSA % than adults
 - Paediatric Specific Burns chart or a Lund & Browder chart must be used

- **Lund & Browder Chart**
 - Most accurate measure tool
 - Can be difficult and time consuming if not familiar
 - Allows for estimation of burn TBSA % in both adults and children



Figure 4.1.1..Chest and Leg escharotomy

Q6. Does a patient with burns require IV FLUID, and if so how much?

- Due to increased vascular permeability and oedema formation secondary to burns patients with large burns are at risk of hypovolaemia and subsequent shock.
- Use of resuscitation fluids is recommended if:
 - **Adult: > 15 – 20% Total BSA**
 - **Children: > 10% Total BSA**
- Other scenarios in which fluid resuscitation may be required include:
 - Electrical burns
 - Coexistent traumatic injuries
 - Delayed presentation
 - Inhalation injury
- An estimation of fluid requirement can be made using the **Modified Parkland formula**. This formula estimates the amount of fluid required for the first 24 hours' post burn.
- Total Fluid Estimation for first 24 hours' post burn = **3 – 4mls x TBSA % Burn x Weight (kg)**
 - **1/2 Total Fluid Volume to be given in first 8 hours**
 - **1/2 Total Fluid Volume to be given over next 16 hours**

Handy tips:

- **Hartmann's** should be considered first line fluid.
- *The timeframe for resuscitation of the initial 8-hour period, and subsequent 16-hour period is taken from the time of the burn, NOT the time of presentation.*
- Patients requiring resuscitation fluids should have an **urinary catheter** placed to allow titration of fluids as the formula provides only an estimate. **Aim for 0.5 ml/kg/hr in adults and 1 ml/kg/hr in children.**
- In any patient who requires burns resuscitation fluids early discussion with regional burns unit is advised for clarification on local policy and preferences.

Q7. What are the specific things to consider in the history of the major burns patient?

- Taking an 'AMPLE' history is just as important in the burns patient as any other trauma patient
- Take special care regarding the events relating to the burn, particularly:
 - The time at which the burn occurred (needed to plan fluid resuscitation)
 - Duration of exposure (prognosis of burns depth)
 - Where they in an enclosed space? (risk of Inhalation injury)

CHAPTER 2. COMPARTMENT SYNDROME

BACKGROUND

- Musculoskeletal compartment syndrome is a limb threatening condition resulting from increased pressure within a muscular compartment, which causes compression of the nerves, muscles and vessels within the compartment.

CAUSES

- **Fractures** (about 75% of cases):
 - Especially tibia, humeral shaft, combined radius and ulna fractures, and supracondylar fractures in children. May be open or closed
- **Soft tissue injuries** due to:
 - Crush injury/ Snake bite/ Excessive exertion/ Prolonged immobilisation
 - Constrictive dressings and plaster casts/ Soft tissue infection/ Seizures
 - Extravasation of intravenous fluids and medications/ Burns/ Tourniquets
- Patients with a **coagulopathy** are at particular risk of compartment syndrome.

COMPLICATIONS INCLUDE:

- **Gangrene** or loss of limb viability requiring amputation
- **Volkman's ischemic contracture** and loss of function
- **Rhabdomyolysis** and Renal failure

ASSESSMENT

- **History**
 - Suspect if:
 - One of the fractures listed above is present
 - One of the soft tissue injuries listed above is present (e.g. Crush injury)
 - Patient has a coexistent bleeding disorder or coagulopathy
 - Remember the **6Ps**
 - **Pain:**
 - Out of proportion to the injury
 - Increased with passive stretch of compartment muscles (most specific)
 - Not relieved by analgesia
 - **Pallor**
 - **Paresthesia**
 - **Polar:** cold limb (late finding)
 - **Paralysis** (late finding)
 - **Pulselessness** (late finding)
 - **Pain is the key symptom.** It occurs early, is persistent, tends to be disproportionate compared with the original injury and is not relieved by immobilisation.
 - **Increase pain with passive stretch is the most sensitive clinical exam finding for compartment syndrome.**
- **Examination**
 - Pain is exacerbated by passive stretching, which is the most sensitive sign.
 - The extremity may be swollen and affected compartments may feel tense and tender on palpation.
 - Assess loss of sensation by light touch and two-point discrimination, rather than just pinprick, which is less sensitive.
 - Refer to a surgeon if compartment syndrome is suspected — do not rely on clinical signs — **have a high index of suspicion!**

- Palpable distal pulses and normal capillary refill does **not** exclude compartment syndrome.
- Pulse oximetry is insensitive and is not recommended in the detection of compartment syndrome.

IMAGING

- Imaging has no role in the diagnosis of compartment syndrome, but may show the presence of fractures and soft tissue injuries that are associated with the condition.

MANAGEMENT

● RESUSCITATION

- Attend to any coexistent life threats
- Ensure adequate oxygenation and systemic circulation if compartment syndrome is potentially present

● SPECIFIC TREATMENT

- Arrange immediate **FASCIOTOMY**
- Remove all constrictive dressings (casts, splints)
- Elevate the limb.
- Reassess in 20 minutes
- Consider measurement of compartment pressures. BUT the diagnosis is primarily clinical and if a compartment syndrome is suspected definitive treatment is SURGERY.
- Consider injury specific measures:
 - Relieve flexion of the elbow if the forearm is involved
 - Apply traction for a partially reduced supracondylar fracture
- If there is no relief within 30 minutes, go straight to the operating theatre

● SUPPORTIVE CARE AND MONITORING

- Provide adequate analgesia
- Provide IV hydration to maintain an adequate urine output in case of rhabdomyolysis
- Frequent monitoring of compartments and neurovascular status of the affected limb

● DISPOSITION

- **Urgent surgical referral** (usually an orthopedic surgeon) and transfer to the operating theatre
- Patients require admission for ongoing monitoring

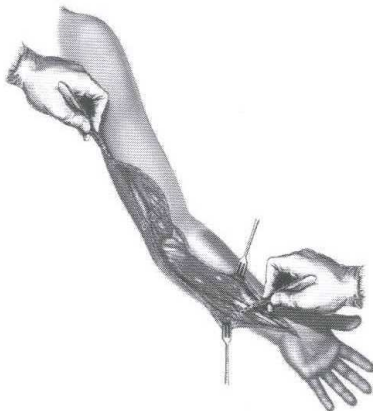


Figure 4.1.2. Compartment fasciotomy

CHAPTER 3. HEAD INJURY

OVERVIEW

- Traumatic brain injury (TBI) is an insult to the brain from an external mechanical force, potentially leading to an altered level of consciousness and permanent or temporary impairment of cognitive, physical, and psychosocial functions.
- TBI accounts for >30% of trauma deaths and is the leading cause of disability in people under 40.
- **Bimodal distribution:**
 - Young adult males
 - Elderly

CAUSES

- **Blunt or Penetrating**
 - Falls (most common cause)
 - MVC (cause of most TBI deaths)
 - Violence and assaults
 - Industrial accidents
 - Sport
- **Consider:**
 - NAI in children
 - Elder abuse
 - Domestic violence

1. PRIMARY HEAD INJURY

- Primary injury occurs at the time of the traumatic incident
- **Mechanisms**
 - **Impact loading:** Collision of the head with a solid object at a tangible speed (contact forces)
 - **Impulsive loading:** Sudden motion without significant physical contact (inertial forces or acceleration/ deceleration injury)
 - **Static loading:** Loading in which the effect of speed of occurrence may not be significant
- Cause brain tissue deformation through:
 - Compression
 - Tension (stretch)
 - Shearing
- Leading to direct cellular and tissue injury:
 - Cell membrane disruption and ion channel dysfunction
 - Blood-brain barrier and vascular disruption
 - Altered autoregulation
 - Local inflammation

2. SECONDARY HEAD INJURY

- Occurs hours to days after the initial insult and is a major determinant of the patients ultimate neurological outcome.
- Attributable to further cellular damage from the effects of primary injuries
- Numerous neurochemical mediators:

- Oxygen free radicals.
- Excitatory amino-acids and endogenous opioid peptides, cytokines and other inflammatory agents.
- Increased metabolism in the injured brain due to increased circulating levels of catecholamines from TBI-induced stimulation of the sympathoadrenomedullary axis and serotonergic system.
- Depression in glucose utilization
- Increase in extracellular potassium may lead to edema
- Decrease in intracellular magnesium may contribute to calcium influx
- Leads to neuronal degeneration and poor outcome
- **Intracranial**
 - Seizure
 - Delayed haematoma or rebleed
 - SAH
 - Vasospasm
 - Hydrocephalus
 - Neuroinfection
- **Systemic**
 - Hypoxia/ Hypotension
 - Hypo/hypercapnia
 - Hyperthermia
 - Hypo/hyperglycaemia
 - Hypo/hypernatraemia
 - Hyperosmolality
 - Infection

GRADING OF HEAD INJURY

- **Mild:** GCS 13-15; 'brief LOC', nausea, cognitive, behavioural and emotional disturbance
- **Moderate:** GCS 9-12 after non-surgical resuscitation
- **Severe:** GCS < 8 after non-surgical resuscitation

CLINICAL FEATURES OF INCREASING ICP

- Vomiting, headache, irritability.
- Seizures.
- Reducing GCS.
- **Cushing's triad**—hypertension, bradycardia, irregular respirations.
- Focal neurology.
- Dilated pupil and contralateral hemiparesis—*uncal herniation causes compression of the 3rd cranial nerve against the tentorium cerebelli, resulting in loss of parasympathetic supply to the ipsilateral eye and unopposed sympathetic activity dilating the pupil; in addition, compression of the corticospinal tract in the midbrain results in contralateral weakness.*

INDICATIONS FOR IMAGING

- CT scanning is the recommended imaging in head injured patients.
- NICE have produced guidance on when CT scanning is indicated.
- Plain X-rays of the skull are not recommended unless as part of a skeletal survey in children presenting with suspected non-accidental injury.
- Consideration should always be made about possible associated neck injuries.

NICE CRITERIA FOR PERFORMING A CT HEAD SCAN

1. ADULTS

- For adults who have sustained a head injury and have any of the following risk factors, **perform a CT head scan within 1 hour of the risk factor being identified:**
 - *GCS less than 13 on initial assessment in the emergency department.*
 - *GCS less than 15 at 2 hours after the injury on assessment in the emergency department.*
 - *Suspected open or depressed skull fracture.*
 - *Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).*
 - *Post-traumatic seizure.*
 - *Focal neurological deficit.*
 - *More than 1 episode of vomiting*
- A provisional written radiology report should be made available within 1 hour of the scan being performed. **[new 2014]**
- For adults with any of the following risk factors **who have experienced some loss of consciousness or amnesia since the injury**, perform a CT head scan within 8 hours of the head injury:
 - *Age 65 years or older.*
 - *Any history of bleeding or clotting disorders.*
 - *Dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs).*
 - *More than 30 minutes' retrograde amnesia of events immediately before the head injury*
- A provisional written radiology report should be made available within 1 hour of the scan being performed. **[new 2014]**

2. CHILDREN

- For children who have sustained a head injury and have any of the following risk factors, **perform a CT head scan within 1 hour of the risk factor being identified:**
 - *Suspicion of non-accidental injury*
 - *Post-traumatic seizure but no history of epilepsy.*
 - *On initial emergency department assessment, GCS less than 14, or for children under 1 year GCS (paediatric) less than 15.*
 - *At 2 hours after the injury, GCS less than 15.*
 - *Suspected open or depressed skull fracture or tense fontanelle.*
 - *Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).*
 - *Focal neurological deficit.*
 - *For children under 1 year, presence of bruise, swelling or laceration of more than 5 cm on the head*
- A provisional written radiology report should be made available within 1 hour of the scan being performed. **[new 2014]**
- For children who have sustained a head injury and have more than 1 of the following risk factors (and none of those in recommendation above), **perform a CT head scan within 1 hour of the risk factors being identified:**

- *Loss of consciousness lasting more than 5 minutes (witnessed).*
- *Abnormal drowsiness.*
- *Three or more discrete episodes of vomiting.*
- *Dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from a height of greater than 3 metres, high-speed injury from a projectile or other object).*
- *Amnesia (antegrade or retrograde) lasting more than 5 minutes.*
- A provisional written radiology report should be made available within 1 hour of the scan being performed. **[new 2014]**
- Children who have sustained a head injury and have **only 1 of the risk factors** in recommendation above should be observed for **a minimum of 4 hours** after the head injury.
- If during observation any of the risk factors below are identified, **perform a CT head scan within 1 hour:**
 - *GCS less than 15.*
 - *Further vomiting.*
 - *A further episode of abnormal drowsiness.*
- A provisional written radiology report should be made available within 1 hour of the scan being performed.
- If none of these risk factors occur during observation, use clinical judgement to determine whether a longer period of observation is needed. **[new 2014]**

PATIENTS HAVING WARFARIN TREATMENT

- For patients (adults and children) who have sustained a head injury with no other indications for a CT head scan and who are having warfarin treatment, **perform a CT head scan within 8 hours of the injury.**
- A provisional written radiology report should be made available within 1 hour of the scan being performed. **[new 2014]**

NICE CRITERIA FOR CERVICAL SPINE IMAGING

1. ADULTS

- For adults who have sustained a head injury and have any of the following risk factors, perform a CT cervical spine scan **within 1 hour** of the risk factor being identified:
 - *GCS less than 13 on initial assessment.*
 - *The patient has been intubated.*
 - *Plain X-rays are technically inadequate (for example, the desired view is unavailable).*
 - *Plain X-rays are suspicious or definitely abnormal.*
 - *A definitive diagnosis of cervical spine injury is needed urgently (for example, before surgery).*
 - *The patient is having other body areas scanned for head injury or multi-region trauma.*
 - The patient is alert and stable, there is clinical suspicion of cervical spine injury and any of the following apply:
 - *Age 65 years or older*
 - *Dangerous mechanism of injury (fall from a height of greater than 1 metre or 5 stairs; axial load to the head, for example, diving; high-speed motor vehicle collision; rollover motor accident; ejection from a motor vehicle; accident involving motorised recreational vehicles; bicycle collision)*
 - *Focal peripheral neurological deficit*
 - *Paraesthesia in the upper or lower limbs.*

- A provisional written radiology report should be made available within 1 hour of the scan being performed. [new 2014]
- For adults who have sustained a head injury and have neck pain or tenderness but no indications for a CT cervical spine scan (see recommendation above), **PERFORM 3-VIEW CERVICAL SPINE X-RAYS WITHIN 1 HOUR** if either of these risk factors are identified:
 - It is not considered safe to assess the range of movement in the neck (see recommendation below).
 - Safe assessment of range of neck movement shows that the patient cannot actively rotate their neck to 45 degrees to the left and right.
 - The X-rays should be reviewed by a clinician trained in their interpretation within 1 hour of being performed. [new 2014]

ASSESSING RANGE OF MOVEMENT IN THE NECK

- Be aware that in adults and children who have sustained a head injury and in whom there is clinical suspicion of cervical spine injury, **range of movement in the neck can be assessed safely before imaging only if no above high-risk factors present and at least 1 of the following low-risk features apply.** The patient:
 - Was involved in a simple rear-end motor vehicle collision
 - Is comfortable in a sitting position in the emergency department
 - Has been ambulatory at any time since injury
 - Has no midline cervical spine tenderness
 - Presents with delayed onset of neck pain. [new 2014]

2. CHILDREN

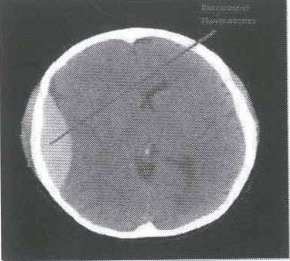
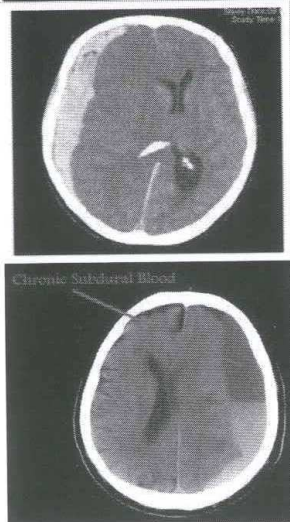
- For children who have sustained a head injury, perform a CT cervical spine scan only if any of the following apply (because of the increased risk to the thyroid gland from ionising radiation and the generally lower risk of significant spinal injury):
 - GCS less than 13 on initial assessment.
 - The patient has been intubated.
 - Focal peripheral neurological signs.
 - Paraesthesia in the upper or lower limbs.
 - A definitive diagnosis of cervical spine injury is needed urgently (for example, before surgery).
 - The patient is having other body areas scanned for head injury or multi-region trauma.
 - There is strong clinical suspicion of injury despite normal X-rays.
 - Plain X-rays are technically difficult or inadequate.
 - Plain X-rays identify a significant bony injury.
- The scan should be performed within 1 hour of the risk factor being identified.
- A provisional written radiology report should be made available within 1 hour of the scan being performed. [new 2014]
- For children who have sustained a head injury and have neck pain or tenderness but no indications for a CT cervical spine scan (see recommendation above), **perform 3-view cervical spine x-rays before assessing range of movement in the neck** if either of these risk factors are identified:
 - Dangerous mechanism of injury (that is, fall from a height of greater than 1 metre or 5 stairs; axial load to the head, for example, diving; high-speed motor vehicle collision; rollover motor accident; ejection from a motor vehicle; accident involving motorised recreational vehicles; bicycle collision).
 - Safe assessment of range of movement in the neck is not possible.



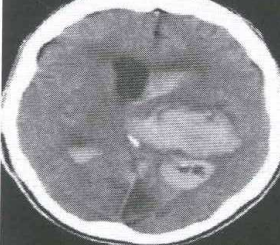
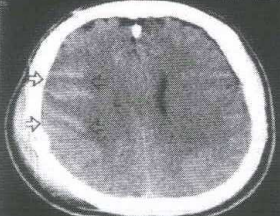
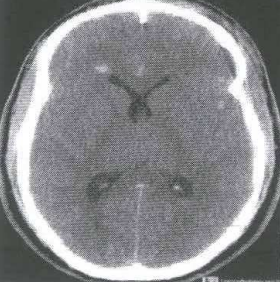
- The X-rays should be carried out within 1 hour of the risk factor being identified and reviewed by a clinician trained in their interpretation within 1 hour of being performed. [new 2014]
- If range of neck movement can be assessed safely in a child who has sustained a head injury and has neck pain or tenderness but no indications for a CT cervical spine scan, **perform 3-view cervical spine X-rays if the child cannot actively rotate their neck 45 degrees to the left and right.**
- The X-rays should be carried out within 1 hour of this being identified and reviewed by a clinician trained in their interpretation within 1 hour of being performed. [new 2014]
- In children who can obey commands and open their mouths, **attempt an odontoid peg view.** [2003, amended 2014]

CT HEAD APPEARANCES

- o **New blood** on CT looks **white**.
- o **Older blood** becomes a similar shade of **grey** to brain tissue.
- o **Air** on a CT scan looks **black** and is the same density as whatever is around the outside of the skull.

Types of intracranial injuries

Extradural or Epidural haemorrhage (EDH)		Biconvex. Cannot cross skull suture lines. Commonly temporo-parietal usually middle meningeal artery . Good prognosis with early treatment. "Lucid" interval in one third of patients—can be minutes or hours.
Subdural haemorrhage (SDH)		Uniconvex. Most common focal lesion. Venous bleed (from bridging dural veins). Can be: Acute: RTA, NAI Chronic: elderly, alcoholics, warfarin, i.e. frequent falls with cerebral atrophy and/ or increased bleeding potential. Acute-on-chronic: acute is whiter; chronic is nearly the same shade of grey as brain tissue. Often able to visualise a line demarcating the two ages of blood.

Subarachnoid haemorrhage (SAH)		<p>Blood in subarachnoid space, i.e. around the brain (blood conforms to sulci and gyri), and in ventricles (wherever CSF goes).</p> <p>Due to tearing of small leptomeningeal arteries and veins.</p> <p>Prognosis better in traumatic rather than spontaneous SAH.</p>
Pneumocephalus		<p>Black 'spots' or patches around edge of the brain.</p> <p>Indicates communication from outside to inside, i.e. fracture of either sinus or skull.</p>
Intracerebral haematoma		<p>Looks white when acute.</p> <p>Size will often cause midline shift (mass effect).</p> <p>Location and size determines neurological signs and treatment.</p> <p>If in posterior fossa, then evacuation more likely to be required.</p>
Cerebral contusion		<p>Small bleeds = white spots on CT.</p> <p>Can be subtle.</p> <p>Often frontal/temporal due to impact of brain tissue with orbital plates or sphenoid ridge.</p> <p>Often associated with SDH.</p>
Diffuse axonal injury		<p>Often very little to see on scans initially. May be pinpoint haemorrhages in corpus callosum and lateral brainstem due to capillary rupture.</p> <p>Widespread, severe white matter injury. Shear strains in acceleration/deceleration injury causes severing of neuronal axons.</p> <p>Commonest cause of a persistent vegetative state.</p>

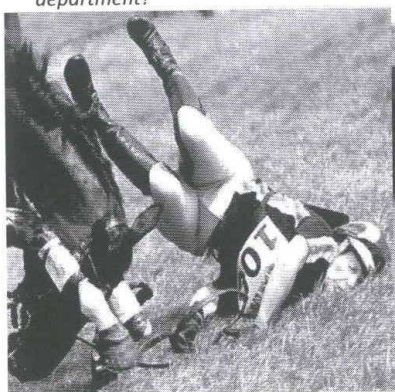
MANAGEMENT PRINCIPLES FOR PREVENTING SECONDARY BRAIN INJURY

- **Primary brain injury** occurs during the initial trauma and results from the displacement of physical structures of the brain. The only way to significantly reduce such injuries is **with accident prevention**.
- **Secondary brain injury** occurs after the initial insult. Many factors are involved in secondary brain injury and are potentially preventable or treatable.
- **CAUSES OF SECONDARY BRAIN INJURY INCLUDE:**
 - Hypoxia and hypercapnea
 - Hypovolaemia and cerebral hypoperfusion
 - Intracranial haematoma with localized pressure effects
 - Increased ICP and cerebral oedema
 - Hyperthermia and seizures
 - Infection.
- The focus of ED management in head-injured patients is the prevention and treatment of secondary brain injury:
 - **Ensure adequate oxygenation** (NICE recommend $\text{PaO}_2 > 13 \text{ kPa}$).
 - **Aim for PaCO_2 in normal range** (NICE recommend PaCO_2 4.5–5 kPa). Intubate and ventilate as required to achieve these aims.
 - **Avoids increases in ICP:**
 - Consider 30° head-up tilt,
 - Avoid cervical collars/ compressions;
 - Tape the ET Tube in place rather than tie it
 - Avoid excessive intra-thoracic pressures.
 - Consider mannitol on specialist advice.
 - **Maintain end organ perfusion** (NICE recommend $\text{MAP} \geq 80 \text{ mmHg}$). Use urine output as indicator of adequate renal perfusion.
 - **Maintain normoglycaemia.**
 - **Treat seizures:** benzodiazepines, prophylactic phenytoin.
 - **Monitor for signs** of neurological deterioration.
 - **Pain management** to avoid increases in ICP.
 - **Temperature control:** aim for normothermia.
 - **Infection control:** wound management; consider need for tetanus booster/immunoglobulin and antibiotics.
- **INDICATIONS FOR REFERRAL TO/DISCUSSION WITH NEUROSURGERY**
 - 'New, surgically significant abnormalities on imaging'.
 - *Persisting coma ($\text{GCS} \leq 8$) after initial resuscitation.*
 - *Unexplained confusion >4 hours.*
 - *Deterioration in GCS after admission.*
 - *Progressive focal neurology.*
 - *Seizure without full recovery.*
 - *Penetrating injury (definite or suspected).*
 - *CSF leak.*

- **ADMISSION CRITERIA FOR HEAD INJURIES**
 - CT with clinically significant abnormalities.
 - GCS not returned to normal.
 - Awaiting scan.
 - Continued clinical concern (e.g. vomiting).
 - Other ongoing concerns (e.g. intoxication, other injuries, suspected NAI, etc.).
- **RECOMMENDED OBSERVATIONS OF HEAD INJURED PATIENTS**
- The following observation should be recorded:
 - GCS
 - Pupil size and reactivity.
 - Limb movements.
 - Respiratory rate.
 - Heart rate.
 - Blood pressure.
 - Temperature.
 - Oxygen saturations.
- **NICE RECOMMEND THE FOLLOWING FREQUENCY OF OBSERVATIONS:**
 - Half-hourly until GCS 15.
 - Then half-hourly for 2 hours.
 - Then hourly for 4 hours.
 - Then 2-hourly.
- **DISCHARGE ADVICE FOR HEAD INJURIES**
 - Verbal and written advice should be given to all patients discharged following a head injury.
 - Advice should be appropriate to the age and language of the patient/carer.
 - If patients have had a CT scan, they should have follow-up arranged with their GP **within 1 week**.
 - Discharge advice should include symptoms that the patient/carer should observe for and return to the ED if they develop.
 - There should also be a section describing symptoms of post-concussional syndrome and where to get help if these are persistent.
- **DISCHARGE ADVICE FOR HEAD-INJURED PATIENTS**
 - Return to the ED if any of the following develop:
 - Unconsciousness or lack of full consciousness.
 - Any confusion.
 - Any drowsiness that goes on for longer than 1 h when you would normally be wide awake.
 - Any problems understanding or speaking.
 - Any loss of balance or problems walking.
 - Any weakness in one or both arms or legs.
 - Any problems with your eyesight.
 - Very painful headache that won't go away.
 - Any vomiting.
 - Any fits (collapsing or passing out suddenly).
 - Clear fluid coming out of your ear or nose.
 - Bleeding from one or both ears and New deafness in one or both ears.

CHAPTER 4. SPINAL TRAUMA

- A 44-year-old woman is brought into a trauma bay in your emergency department after a fall from a horse. She has a hard collar on and her head is taped to the hard spine board she rode in on. It is immediately apparent that although she is GCS 15, she is unable to move any of her limbs.
- Are you up to the task of assessing and managing spinal trauma in the emergency department?

**Q1. Which major trauma patients should have c-spine precautions?**

- Those with:
 - Neck pain or neurological symptoms
 - Altered level of consciousness
 - Significant blunt injury above the level of the clavicles

Q2. How is the cervical spine immobilized?

- A **hard collar** is applied, but by itself this inadequately immobilizes the cervical spine.
- The main purpose of a cervical spine collar is to remind healthcare workers that the neck has not yet been cleared.
- Adequate immobilisation requires the addition of either:
 - **Manual immobilization**
 - **Application of sand bags and head tape**
- Appropriate cervical spine precautions is an evolving area of controversy...

Q3. The patient says she feels nauseated and is worried she might vomit. What should you do if a patient in cervical spine precautions starts to vomit?

- **Immediate intervention is needed:**
 - **Log roll** if 4 people are available: one to hold the head, and three to perform the roll
 - If insufficient numbers of people are available, **tilt the bed head down** so that vomit runs clear of the airway; **Suction** away the vomitus with a Yankauer.
- **Further intervention:**
 - Administer **antiemetics** (e.g. ondansetron 4mg IV)
 - **Seek and treat underlying causes:** patients often vomit due to pain or opioid analgesia, but it can also be a sign of worrying causes such as serious abdominal trauma, hypotension or raised intracranial pressure

Q4. What is the difference between primary and secondary spinal cord injury?

- **Primary spinal cord injury**
 - Results from blunt or penetrating mechanisms at the time of the initial traumatic event. Fractures, dislocations, hematomas and soft tissue swelling directly injury the spinal cord via mass effect, disrupted blood supply or transection.
- **Secondary spinal cord injury**
 - Occurs after primary spinal cord injury due to mechanical instability contributing to ongoing direct injury, or insults from other factors such as hypoxia and hypoperfusion.
 - The later may be due to associated injuries in a multiple injured patient or due to spinal cord injury associated respiratory insufficiency and neurogenic shock.
 - Much of the acute management of spinal cord injury is aimed at preventing secondary spinal cord injury.

Q5. You notice that the patient is now mildly tachypneic with shallow breaths and a weak cough. How do patients with spinal injuries develop respiratory insufficiency?

- The mechanism of respiratory insufficiency varies according to the level of injury:
 - **High cervical injuries** may lead to airway obstruction due to local hematoma and swelling.
 - **Lesions at the C5 level or higher** lead to diaphragmatic paresis or paralysis, as the phrenic nerve arises from the C3-5 levels.
 - **Thoracic or higher lesions** may lead to respiratory distress due to paralysis of intercostal muscles, as the intercostal nerves arise from the T1-12 levels.
- Other causes include:
 - Coexistent thoracic injuries
 - Coexistent traumatic brain injury (e.g. decreased respiratory drive from coma)
 - Complications of spinal cord injury (e.g. aspiration, atelectasis, pulmonary embolus, metabolic acidosis from spinal shock)
 - Complications of treatment (e.g. sedation, fluid overload, transfusion-associated acute lung injury, ventilator associated pneumonia).
- Observe spinal injury patients closely for evidence of respiratory insufficiency — early signs include mild tachypnea with shallow breaths and a weakening cough.

Q6. The patient's vital signs are rechecked. She has a blood pressure of 80/50 mmHg.

What is the most likely cause of hypotension in a major trauma patient with a possible spinal cord injury?

- **Haemorrhage**
 - Any hypotension in a trauma patient should be assumed to be hypovolaemic in origin until proven otherwise, even in a patient with an overt spinal injury.
 - Sources of bleeding must be aggressively sought and controlled.
- **Neurogenic shock** is rare in comparison.

Q7. No source of bleeding can be found on physical examination or on the extended FAST scan performed at the bedside. Her heart rate is only 50/min.

The medical student attached to your team asks if this means the patient has spinal shock.

What is the difference between neurogenic shock and spinal shock?

- The patient's vital signs are consistent with **neurogenic shock**.
 - **Neurogenic shock** is classically characterised by hypotension, bradycardia and peripheral vasodilatation. Neurogenic shock is due to loss of sympathetic vascular tone and happens only after a significant proportion of the sympathetic nervous system has been damaged — as may occur with lesions at the T6 level or higher.

- **Spinal shock** is not a true form of shock. It refers to the flaccid areflexia that may occur after spinal cord injury, and may last hours to weeks. It may be thought of as 'concussion' of the spinal cord and resolves as soft tissue swelling improves. Priapism may be present.

Q8. Describe your overall approach to the management of a patient with spinal cord injury.

- **Activate the trauma team** and use a coordinated team-based approach in a dedicated trauma bay appropriately staffed and equipped for resuscitation. Remove the patient from a rigid spine board as soon as possible by transferring onto a trauma bed.

A. Primary survey and resuscitation (ABCDE approach)

- **Airway maintenance with cervical spine immobilization**
 - Lesions above C5 require intubation for mechanical ventilation
 - Maintain cervical spine precautions (hard collar, sandbags, tape forehead)
- **Breathing and ventilation**
 - High flow oxygen 15L/min via a non-rebreather mask
 - Monitor for respiratory insufficiency from thoracic or higher spinal lesions, complications of spinal cord injury, or other thoracic injuries
- **Circulation with haemorrhage control**
 - Look for neurogenic shock, which may also mask hemorrhagic shock (which is more common)
 - Treat neurogenic shock with repeated fluid boluses (e.g. 250 mL crystalloid) +/- noradrenaline infusion to maintain organ perfusion (normal mentation, warm peripheries, urine output >0.5 mL/kg/h) and/or a MAP >65 mmHg.
 - Be aware that vagal stimuli (e.g. suction, NGT insertion, intubation) may exacerbate neurogenic shock; treat with **atropine 0.6mg IV boluses**.
- **Disability (neurological evaluation)**
 - Detailed neurological exam including motor and sensory levels bilaterally; check for priapism; check anal sphincter tone and bulbocavernosus reflex.
- **Exposure and Environmental Control**
 - Higher risk of hypothermia due to peripheral vasodilation resulting from sympatholysis and decreased muscular activity
 - Use fluid warmer, warm blankets, and/or bair hugger to keep patient warm

Adjuncts to Primary Survey and Resuscitation

- **ECG** and full non-invasive monitoring including temperature
- **Obtain trauma series radiographs** (lateral cervical spine XR, chest XR, pelvic XR)
- **Bedside ultrasound** to identify other injuries and sources of haemorrhage (e.g. EFAST)
- **Nasogastric tube insertion** — spinal patients are at risk of gastric distention
- **Indwelling catheter insertion** — prevent urinary retention and bladder overdistention due to spinal injury; monitor urine output given the risk of hemorrhagic and neurogenic shock.
- **Consider transfer:** Organize early transfer to a spinal unit

B. Secondary survey

- Head-to-toe examination looking for other injuries
- **Adjuncts to Secondary Survey**
 - **Organize imaging** to define the nature of the spinal cord injury (CT is more readily available and defines bony injuries; MRI defines neurological injury)
 - **Monitor for fluid overload** using bedside ultrasound (look for IVC distention and pulmonary edema) and/or CVP monitoring.

- **Continued post-resuscitation care and monitoring**
 - Remember **FASTHUGS IN BED Please!** (see below)
- **Definitive care and disposition**
 - Transfer to spinal unit for ongoing care.

Q9. How is spinal cord injury classified and how the level of spinal injury determined?

- Use the **American Spinal Injury Association (ASIA)** classification to avoid confusion.
- The ASIA classification is based on:
 - **The neurological level** — the most caudal segment with normal function
 - **Severity of the neurological deficit** — graded A to E on the ASIA Impairment scale (A is complete, E is normal) based on degree of intact motor and sensory function.
 - Spinal cord injury patients require careful examination and documentation of the level initially, and this should be performed serially. This allows early detection of injury progression or of resolution of spinal shock (bulbocavernosus reflex typically returns after about 48 hours unless there is a complete spinal cord injury).

ASIA/ISCOS EXAM CHART (ASIA IMPAIRMENT SCALE)

Grade A	Complete lack of motor and sensory function below the level of injury (including the anal area)
Grade B	Some sensation below the level of the injury (including anal sensation)
Grade C	Some muscle movement is spared below the level of injury, but 50 percent of the muscles below the level of injury cannot move against gravity.
Grade D	Most (more than 50 percent) of the muscles that are spared below the level of injury are strong enough to move against gravity.
Grade E	All neurologic function has returned.

Q10. How are the thoracic and lumbar spine cleared in major trauma?

- There are no validated decisions rules for clearing the thoracic or lumbar spine.
- Imaging is generally required if there are the following:
 - *Point tenderness*
 - *Deformity or bony step*
 - *Neurological findings consistent with a thoracic or lumbar injury*
 - *High risk mechanism, especially in the presence of distracting injuries*
- Note that significant thoracolumbar spinal injury can be present in the absence of midline tenderness.
- ‘**FAST HUGS IN BED Please**’, which applies equally well in the emergency department or the intensive care unit:
 - *Fluid therapy And Feeding*
 - *Analgesia, Antiemetics and ADT (AAA)*
 - *Sedation and Spontaneous breathing trial*
 - *Thromboprophylaxis*
 - *Head up position (30 degrees) if intubated*
 - *Ulcer prophylaxis*
 - *Glucose control*
 - *Skin/ eye care and Suctioning*
 - *Indwelling catheter*
 - *Nasogastric tube*
 - *Bowel cares*
 - *Environment (e.g. temperature control, appropriate surroundings in delirium)*
 - *De-escalation (e.g. end of life issues, treatments no longer needed)*
 - *Psychosocial support (for patient, family and staff)*

SPINAL CORD SYNDROMES

SYNDROMES		CAUSES	CLINICAL FINDINGS
Complete transection	cord	<ul style="list-style-type: none"> • Trauma, • Infarction, • Transverse myelitis • Abscess, • Tumour 	<ul style="list-style-type: none"> • Complete loss of sensation below level • Complete paralysis below level
Cord hemisection/ Brown-sequard syndrome		<ul style="list-style-type: none"> • Trauma, • MS • Abscess, • Tumour 	<ul style="list-style-type: none"> • Ipsilateral loss of motor and proprioception • Contralateral loss of pain and temperature
Central syndrome	cord	<ul style="list-style-type: none"> • Neck hyperextension • Spinal stenosis • Syringomyelia • Tumour 	<ul style="list-style-type: none"> • Motor impairment > sensory • UL>LL • Distal > proximal • Bladder dysfunction
Anterior syndrome	cord	<ul style="list-style-type: none"> • Hyperflexion • Disc protusion • Anterior spinal artery occlusion • Post AAA 	<ul style="list-style-type: none"> • Motor function impairment • Pain and temperature loss • Proprioception spared
Cauda equina syndrome	equina	<ul style="list-style-type: none"> • Disc prolapse • Tumour • Infection 	<ul style="list-style-type: none"> • Bladder/bowel dysfunction • Saddle anaesthesia • Sexual dysfunction

BROWN-SÉQUARD SYNDROME

- Refers to a **hemisection** (one sided lesion) of the spinal cord. This is most often due to traumatic injury, and involves both the anterolateral system and the DCML pathway:
- DCML pathway: **ipsilateral loss of tactile sensation and proprioception**
- Anterolateral system: **contralateral loss of pain and temperature sensation**.
- It will also involve the descending motor tracts, causing **ipsilateral hemiparesis**.

CHAPTER 5. THORACIC TRAUMA

1. TENSION PNEUMOTHORAX

Definition and context

- Think expanding pneumothorax that increasingly limits ventilation and venous return — perhaps a better concept for teaching purposes. It is not an on/off phenomenon, rather a continuum. So even impressive expansion may be well tolerated in young individuals with no co-morbidities and no other injuries.
- In fact tolerated so well that you may miss the clinical diagnosis — no harm done.
- Given that the expansion is dynamic, be vigilant in patients with a chest x-ray proven small pneumothorax in whom you elect not to insert a chest drain.

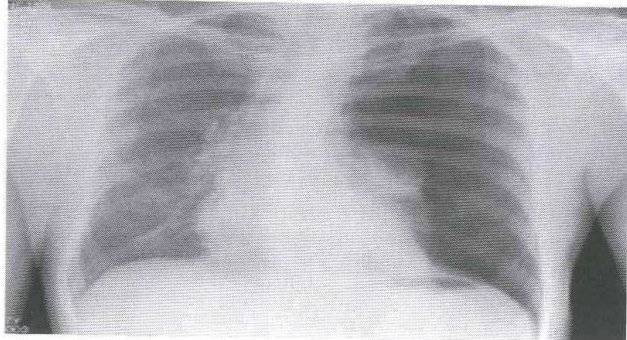


Fig 4.5.1. Tension pneumothorax

Clinical assessment and identification

- Symptoms and signs depend on where your patient is on the expanding pneumothorax continuum — clinical features become more obvious with expansion.

a. For awake patients:

- Universal features of tension pneumothorax are **chest pain** and **respiratory compromise**, neither of which are discriminatory of course.
- **Low oxygen saturations** may be an early feature; **hypotension** tends to be late. Both may have other causes.
- Lateralising the pneumothorax may not be straightforward — listen for **decreased breath sounds on the affected side**. Listen in the axillae rather than over the anterior chest wall.
- Note the classical signs of **hyper-resonance** and **tracheal deviation** are soft and difficult to elicit.

b. For ventilated patients:

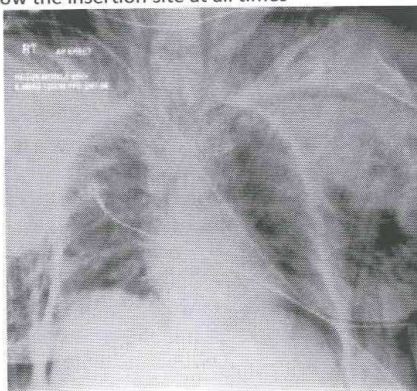
- Early reliable signs are:
 - **↓SPO₂, ↓BP, ↑HR, ↑VP**
 - Decrease in oxygen saturations — this is likely to be prompt
 - Decrease in BP, Tachycardia
 - Look too for raised ventilation pressure (greater than 40) — ensure that the ventilator pressure alarm settings are set appropriately.
 - **Lateralising signs** are the same as for awake patients.
- A portable CXR is recommended for tension pneumothorax, unless the patient is critical
- Radiological evidence of tensioning does not necessarily correlate clinically

ED MANAGEMENT OF TENSION PNEUMOTHORAX

- **Needle thoracocentesis** is advocated for tension pneumothorax in the first instance in the ATLS manual. Potential drawbacks to this strategy are:
 - **It tends to get over used**, particularly in stable resus room patients in whom portable CXR is readily available and chest drain is the preferred treatment.
 - **A lack of hiss** (or bubbling, if you have put some saline in a syringe attached to the needle) might be considered as evidence of no tension pneumothorax – the procedure doesn't have 100% sensitivity.
 - **Three potential drawbacks to the recommendation of using needle thoracosynthesis:**
 - *A (4.5 cm) 14-gauge cannula may not reach the pleural space via the second intercostal space.*
 - *The cannula can kink and cease to function*
 - *A pneumothorax may be caused if the diagnosis is incorrect.*
- **Thoracostomy**
 - Avoid needle thoracocentesis in peri-arrest patients with suspected tension pneumothorax – thoracostomy is the better option
- **Chest drain insertion**
 - The most common cause of serious injury (and death) as a result of chest drain insertion, is insertion at the incorrect site, usually too low
 - Confirm that the drain lies within the chest wall cavity by looking for fogging of the tube and swinging of the chest drain with respiration.
 - Do not clamp the chest drain or apply suction
 - The underwater seal needs to remain below the insertion site at all times

Key learning Points

- If you do perform needle thoracocentesis, have some saline in the syringe to demonstrate bubbling when the tension is hit
- Gross surgical emphysema with pneumomediastinum (as per CXR) and a chest drain that continues to bubble, suggests **tracheo-bronchial injury**.
- If there is good clinical and radiological evidence of significant lateral chest wall injury, consider the second intercostal space anteriorly for the chest drain insertion – it's safer for the operator and less painful for the awake patient.
- One third of initial CXRs in trauma will not detect pneumothorax; anaesthetic colleagues need to be aware of this if your patient leaves for theatre.
- Cardiac tamponade may give similar signs clinically shock, with distended neck veins.
- A combination of your FAST skills, urgent CXR and consideration of the mechanism of injury should help you distinguish the two.
- Beware other pathology masquerading as large (possibly tensioning?) pneumothorax on the CXR, for example an emphysematous bulla or gastrothorax.
- Reconsider the clinical presentation and consider CT where the CXR diagnosis remains in doubt

**Figure 4.5.2. Tracheo-bronchial injury**

2. OPEN PNEUMOTHORAX

Definition and context

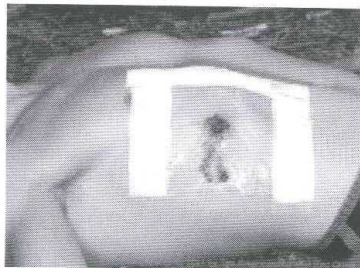
- Think hole in the chest. It is also known as a **communicating pneumothorax** or **sucking chest wound**.
- A hole of only 1 or 2 cm in radius may cause serious respiratory compromise, particularly in patients with comorbidities, and/or other injuries
- Rarely, it is caused by ballistic (shot gun) injury. Clearly, this unlikely to be missed clinically.
- As the patient takes a breath in, the hole in the chest competes with the normal airway (mouth/nose to trachea) for delivery of air.

Clinical assessment and identification

- Prompt clinical inspection front and back; a small sucking chest wound is usually audible.

Treatment

- The emergency physician must alleviate any respiratory embarrassment, exclude associated injuries and identify the need for timely **thoracotomy or laparotomy**.
- **Cover the wound with a 3-sided sterile occlusive dressing**, if not already done so by the paramedics.
- Temporarily release any wound dressing over the open pneumothorax if you suspect tensioning
- **Early intubation:** IPPV solves the respiratory embarrassment created by the hole in the chest
- For small open pneumothoraces, insert a **chest drain** remote from the wound on that side; this is practically easier once the patient is anaesthetised
- *Do not insert a chest drain in patients with a large open pneumothorax since muscle flaps may be needed for closure and can be damaged in the procedure*
- Definitive treatment is surgical repair.



3. MASSIVE HAEMOTHORAX

Definition and context

- Massive Haemothorax is a haemothorax with a **volume greater than 1500 ml**, or greater than **one third of the patient's blood volume**.
- This is an uncommon injury which can be caused by blunt or penetrating trauma, and is unlikely to be missed radiologically.
- It creates a problem because of shock (haemorrhagic and impaired venous return from the vena cava) and decreased ventilation (the lung on that side gets compressed).

Clinical assessment and identification

- Think of the concept of expanding haemothorax (another continuum!): the signs will be less reliable in moderate haemothorax.
- Listen at the lung bases (Figure below).
- There should be clear signs of shock prompting you to rule out the diagnosis.
- Use CXR and FAST to guide you.
- You may underestimate the size of the haemothorax on a supine CXR (Figure below).

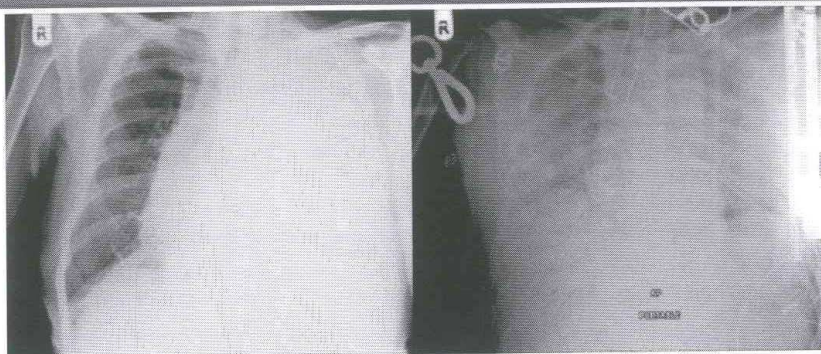


Figure 4.5.3. Lung bases and Massive haemothorax on supine CXR

- **FAST signs:**

- The absence of a mirror image of liver/ lung or spleen/lung across the diaphragm suggests a haemothorax;
- Alternatively, free fluid in the abdomen alone should prompt you to reconsider the source of haemorrhage.

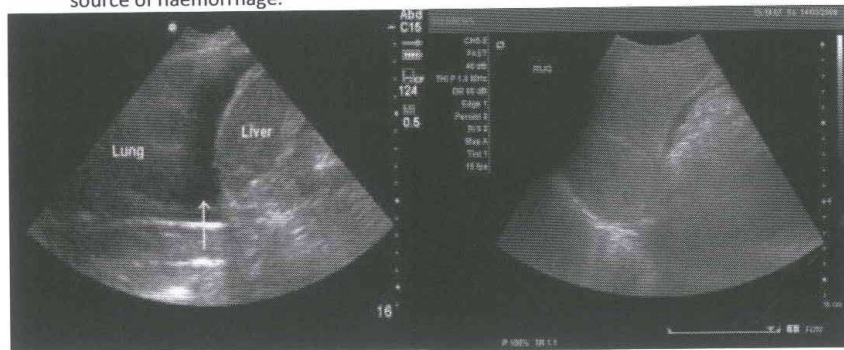


Figure 4.5.4. Right Haemothorax

Figure 4.5.5. Free fluid in the abdomen

Treatment

- **ABCD approach.**
- **Intravenous fluid** resuscitation
- **Blood** and blood products (autotransfusion)
- **Chest drain**
- Consider **early surgical referral**
- In cases of exsanguinating haemorrhage, clamp the chest drain and arrange immediate **thoracotomy in theatre.**

ATLS indications for Thoracotomy

- Prompt drainage of 1500ml blood or a third of the patient's circulating volume
- More than 200ml/hr loss for 2-4 hours
- Continued need for blood transfusion

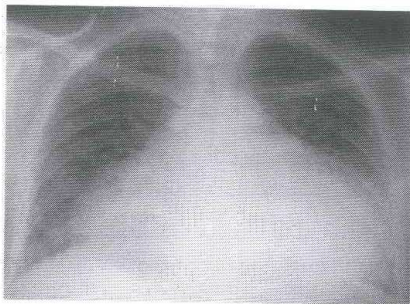
4. CARDIAC TAMPONADE

Definition and context

- Cardiac tamponade is a collection of fluid (blood in the context of trauma) in the pericardial sack causing haemodynamic compromise.

- When faced with a penetrating injury to chest, back or upper abdomen, think tension pneumothorax, think massive haemothorax, and think cardiac tamponade.*

- Exclude or confirm tamponade with a FAST scan.*



- Cardiac tamponade is not an on/off phenomenon (yet another continuum), though the progression to PEA cardiac arrest may be rapid.
- 50 to 200 ml of blood in the pericardial sac may be enough. Cardiac tamponade as a result of blunt injury is exceptionally rare in those patients reaching hospital alive.

Clinical assessment and identification

- FAST has particularly high sensitivity (about 95% according to ATLS).
- Do note that there are drawbacks in detecting and interpreting the classical clinical signs (**Beck's Triad**):
 - Neck veins** may not be distended if the patient has haemorrhagic shock
 - Hypotension** (and a raised respiratory rate) may have other causes
 - Muffled heart sounds** unlikely to be heard in the ED!

Treatment

- ABCD approach with Fluid resuscitation** to increase pre-load
- If the patient is **haemodynamically stable** refer for **urgent surgical exploration** in theatre.
- Look for co-existing injuries (especially pneumothorax) on a portable CXR first
- Thoracotomy** if the patient presents within 10 minutes of cardiac arrest
- Correctly performed **pericardiocentesis** is likely to fail because the blood within the pericardium is clotted. The procedure will also delay thoracotomy.
- ATLS advises pericardiocentesis only as a temporising measure, pending thoracotomy.

5. FLAIL CHEST

Definition and context

- This occurs when a series of ribs (usually 3 or more) are fractured segmentally (i.e. in more than one place) resulting in a free or floating section of the chest wall.
- This injury is relatively common – small flails may be missed clinically.
- Beware **underlying pulmonary contusions** which are inevitable, and may cause significant morbidity and mortality in any age group
- Considerable force is required to create a flail chest in young people look carefully for other injuries, both intra and extra-thoracic.
- Multiple rib fractures are a potential source of significant haemorrhage.

Clinical assessment and identification

- By palpation as well as inspection.
- A CXR might identify associated pneumothorax, haemothorax and pulmonary contusions.

- The appearance of early pulmonary contusions is particularly worrying; evidence of further and perhaps extensive contusion (with physiological effect) may evolve.

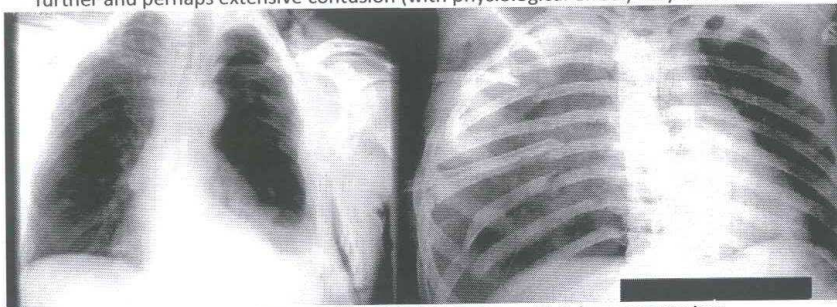


Figure 4.5.6. Multiple Ribs fractures with Pulmonary contusions

Treatment

- Treatment options depend largely on the respiratory embarrassment caused: consider your patient's clinical condition, the size of the flail chest, associated injuries, age, co-morbidities and destination from resus (theatre, CT scan, ITU or ward)
- **For patients with major trauma (Life-threatening):**
 - **Intubation and ventilation (IPPV).** This enables you to take better control of respiratory compromise,
 - **Pain management** (remember to give **adequate morphine post RSI**) and facilitates clinical procedures e.g. chest drain insertion and CT scan
 - Insert a **chest drain** for associated pneumothorax and haemothorax.
 - **CT** is likely to pick up occult pneumothoraces; whilst usually small, chest drain insertion is recommended if the treatment option is ventilation
 - Judicious **fluid resuscitation** since excessive fluid floods injured lung tissue
 - **Definitive surgery** (internal fixation of ribs) at the discretion of cardiothoracic surgeons.
- **If no life-threatening injuries:**
 - Discuss treatment options with **ICU and thoracic surgical** colleagues for patients with a flail segment causing limited respiratory embarrassment.
 - A **conservative approach** might include the use of **thoracic epidural, intercostal nerve blocks** or patient controlled analgesia, and **CPAP** and **Physiotherapy**.

6. PULMONARY CONTUSION

Definition and context

- Bruised lung; unlikely to be missed radiologically unless the CXR is early.
- Potentially life threatening since:
 - The patient is at **risk of hypoxaemia**
 - Because of the force involved to cause the injury, **associated injuries are common**
 - Injured lung is **vulnerable to flooding** from aggressive fluid resuscitation
 - Patients with co-morbidities and/or advanced age are particularly at risk from this injury.

Clinical assessment and identification

- Look for patchy white areas progressing to frank consolidation on the CXR (aspiration and haemorrhage are differential diagnoses)

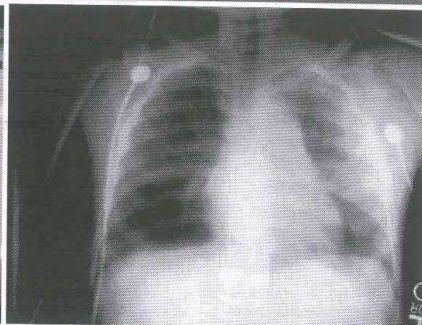
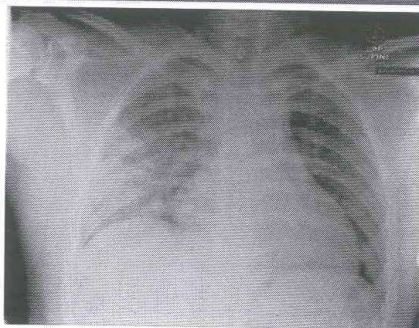


Figure 4.5.7. Lung contusion

- Contusions visible on the initial CXR suggests significant injury, with further radiological changes and blood gas derangement likely to follow.
- Look for associated rib fractures and haemo/pneumothorax
- Rib fractures do not always co-exist, particularly in the young (where their existence indicates that significant force created the injury)

Treatment

- **A&B: IPPV with Positive End Expiratory Pressure (PEEP)** for the sicker patients
- **C: Judicious use of fluids**
 - Consider insertion of a central line and arterial line
 - **Avoid colloids** since these will breach injured lung tissue and worsen hypoxia
- **D:** No evidence for steroids or prophylactic antibiotics
- Discuss disposition of each patient with **ITU and thoracic surgical colleagues**

7. MYOCARDIAL CONTUSION

Definition and context

- Myocardial bruising caused by blunt injury, including deceleration and ballistic mechanisms.
- The key problem with interpreting the literature is the lack of a diagnostic gold standard (apart from post mortem).

Clinical assessment and identification

- A normal ECG effectively rules out the condition.
- Unexplained tachycardia may be a clue. Look too for atrial and ventricular ectopics.
- Consider bedside echocardiogram.
- Consider troponin.
- Beware labelling ST changes as myocardial contusion; there may have been a primary cardiac event that precipitated the accident.

Treatment

- There is no direct ED-based intervention to treat the myocardial contusion itself; treat the following if identified:
 - Hypoxaemia
 - Acidaemia
 - Fluid status
 - Low haemoglobin
- Monitor ECG. Consider a central and arterial line.

8. DIAPHRAGMATIC INJURY

Definition and context

- Diaphragmatic injury is usually caused by penetrating rather than blunt injury.
- It is easily missed both clinically and radiologically.
- In blunt injury it is three times more common on the left (the right hemi-diaphragm being protected by the liver) and nearly always at the weakest point, posterolaterally.
- A diaphragmatic breach will not heal spontaneously because of the differential pressure gradients between chest and abdomen.
- Abdominal content herniation is a possibility and may be picked up years later.

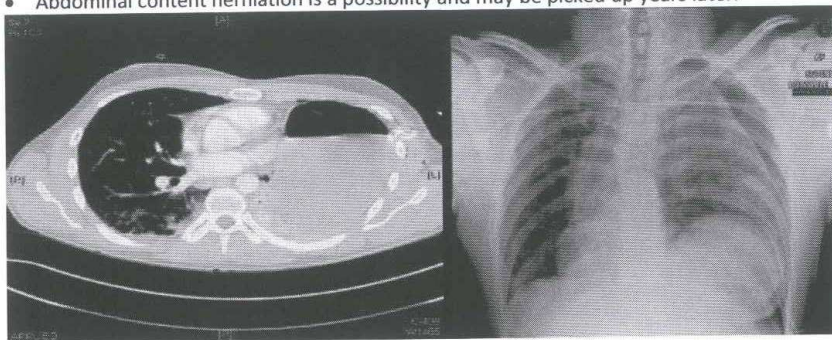


Figure 4.5.8. Diaphragmatic injury

Clinical assessment and identification

- Symptoms are likely to be masked by associated injuries.
- Diaphragm injuries resulting from knives or bullets are more likely to be detected on surgical exploration.
- In blunt injuries, particularly those causing an abrupt rise in intra-abdominal pressure, be careful not to interpret a gastrothorax for a large pneumothorax; both will cause respiratory embarrassment.

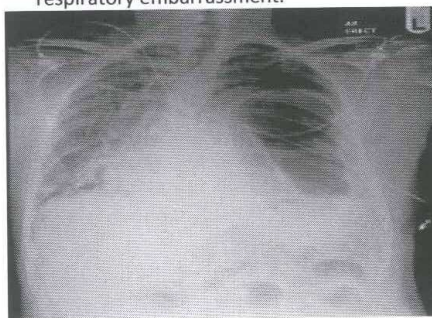


Figure 4.5.9. Large gastrothorax

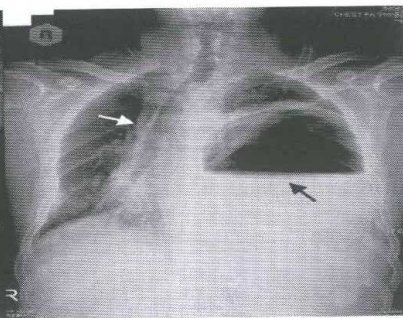


Figure 4.5.10. Tension gastrothorax

Treatment

- **Insert a nasogastric tube** gently to drain stomach content.
- A cautiously placed **chest drain** using the traditional open technique, not Seldinger, is indicated.
- **Surgical repair** needs to be considered in the context of associated injuries.

9. OESOPHAGEAL INJURY

- This rare injury is often initially missed both clinically and radiologically.
- Other associated injuries will normally predominate the clinical presentation e.g. a neck stabbing with tracheal and vascular disruption.
- Operative repair or endoluminal stenting should be considered in the context of other associated injuries.

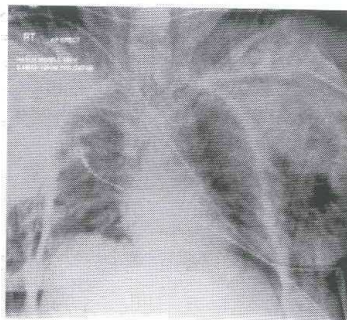
10. TRACHEAL/BRONCHIAL INJURY

Definition and context

- This rare injury is typically caused by significant deceleration injuries; most patients die at the scene of the accident.
- It is unlikely to be missed clinically or radiologically in survivors, since clinical effects are usually dramatic.

Clinical assessment and identification

- A massive air leak is suggested by **gross surgical emphysema**, **pneumomediastinum** and a **vigorously bubbling chest drain** that has failed to alleviate respiratory compromise.
- **Haemoptysis** is an additional clue.



Treatment

- Discuss **intubation strategy** with senior anaesthetic colleagues (consider single or double cuffed tubes, use of fibre optics, etc).
- Consider **additional large bore chest drain** on the affected side (one intercostal space further up). *Do not attach suction to the chest drain.*
- Other significant patient injuries may influence your resuscitation strategy.

11. SIMPLE PNEUMOTHORAX

- This is a common injury which is readily missed on CXR and subsequently discovered on CT.
- Small, asymptomatic/occult pneumothoraces may be observed, even if the patient is ventilated.
- About a third may deteriorate clinically, necessitating a drain.
- No guideline regarding the safe timing for flying following a simple traumatic pneumothorax exists.
- A pragmatic approach may be to adopt British Thoracic Society guidelines for spontaneous pneumothorax: *flying is permissible, once chest x-ray confirms resolution of the pneumothorax.*

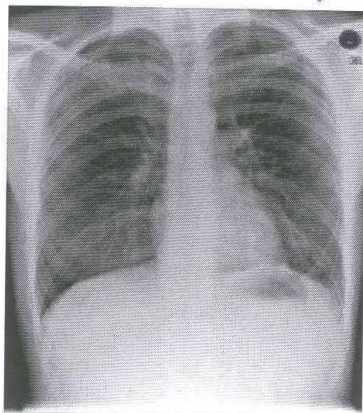


Figure 4.5.11. Simple pneumothorax

12. RIB FRACTURES

- Significant force is required to break ribs in the young; underlying injury is typical, especially lung contusions.
- Whilst less force is required in the elderly, even an isolated rib fracture can result in significant morbidity (e.g. secondary pneumonia) particularly in those with pre-existing comorbidities.
- In addition to standard therapy consider the role of patient-controlled **analgesia**, **thoracic epidural** and **physiotherapy** for vulnerable patients.

13. STERNAL FRACTURES

- These are relatively benign injuries but may be associated with underlying myocardial or pulmonary contusion.
- Consider the role of patient-controlled **analgesia** or **local anaesthetic** via a sternal catheter in vulnerable patients.



Figure 4.5.12. Sternal fracture

14. POSTERIOR STERNOCLAVICULAR JOINT DISLOCATION

- This is an exceptionally rare injury. It is clinically important since the medial clavicular head may compromise the airway or major vessels.
- If there is evidence of compromise, reduction of the dislocation should be attempted.
- Abduct the arm to 90° and extend 10-15° and apply traction (with counter attraction to the torso from another colleague); maintain traction and pull the medial end of the clavicle forward with your fingers and thumb.
- If this fails, prepare the skin with iodine and local anaesthetic and repeat with a towel clip.

CHAPTER 6. ACUTE AORTIC DISSECTION

DEFINITION

- **Aortic dissection** occurs following a tear in the aortic intima with subsequent separation of the tissue within the weakened media by the propagation of blood.
- **A ruptured or leaking abdominal aneurysm** is a different disease, requires immediate surgery with only occasional need for any imaging, can be performed in most hospitals by a vascular surgeon and does not require the use of cardiopulmonary bypass.
- The most common catastrophe of the aorta (3:100,000); 3 times more common than AAA rupture

CLASSIFICATION

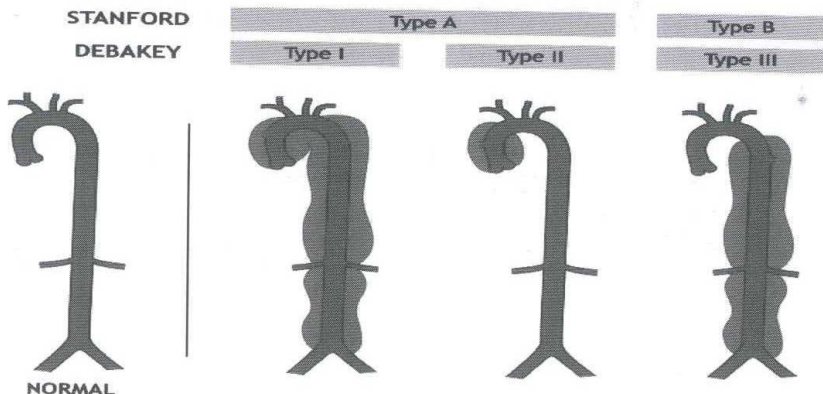
Stanford classification

- There are 4 different classifications of aortic dissection and the commonest one used is the **Stanford classification** dividing them into **Type A** and **Type B**.
 - **Type A** dissection involves the Ascending Aorta
 - **Type B** dissections involve only the Descending Aorta and occur distal to the origin of the left subclavian artery.

DeBakey's Classification

- **Type I** dissections involve the entire aorta whilst
- **Type II** only involves the ascending aorta and, or the arch of the aorta.
- **Type III** involves only the descending aorta.

- **Reul and Cooley** further subdivided De Bakeys classification into subtypes **IIla** and **IIlb**.
 - In **IIla** the dissection involves the aorta just distal to the left subclavian artery but extends proximal or distal to this but is largely above the diaphragm.
 - In **IIlb** the dissection occurs only distal to the left subclavian artery and may extend below the diaphragm.



PATHOPHYSIOLOGY

- There are 3 possibilities as to how the blood gets into the media:
 - Atherosclerotic ulcer leading to intimal tear
 - Disruption of vasa vasorum causing intramural haematoma
 - De novo intimal tear

- Following dissection, blood flow into the media may cause:
 - Extension up or down
 - Rupture
 - Vessel branch occlusion
 - Aortic regurgitation
 - Pericardial effusion / tamponade
- 80% of aortic dissections are in non-aneurysmal vessels

• RISK FACTORS

PREDISPOSING FACTORS FOR AORTIC DISSECTION
Hypertension
Ehlers-Danlos Syndrome
Marfan's syndrome
Turners Syndrome
Pregnancy
Cocaine abuse
Coarctation of the aorta
Bicuspid aortic valve
Annuloaortic ectasia and familial aortic dissection
Giant cell arteritis
Iatrogenic

HISTORY

- Chest pain (ripping, tearing in nature, sudden onset, maximal @ onset) – not always present!
 - Retrosternal chest pain – anterior dissection
 - Interscapular pain – descending aorta
 - Severe pain ('worst ever-pain') (90%)
 - Sudden onset (90%)
 - Sharp (64%) or tearing (50%)
 - Migrating pain (16%)
 - Down the back (46%)
 - Maximal at onset (*not* crescendo build up, as in an AMI)
- Other
 - End-organ symptoms: neurological, syncope, seizure, limb paraesthesias, pain or weakness, flank pain, SOB + haemoptysis
 - Aortic regurgitation
 - Hypertension
 - Most have ischaemic heart disease

EXAMINATION

- Aortic regurgitation is common
- Hypertension (if hypotensive ensure it is not due to limb discrepancy caused by an occluded vessel – check BP in the arm with best radial pulse)
- Shock – ominous signs: tamponade, hypovolaemia, vagal tone
- Heart failure
- Neurological deficits: limb weakness, paraesthesiae, horners syndrome
- SVC syndrome – compression of SVC by aorta
- Asymmetrical pulses (carotid, brachial, femoral)
- Haemothorax

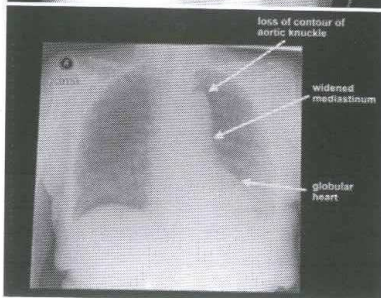
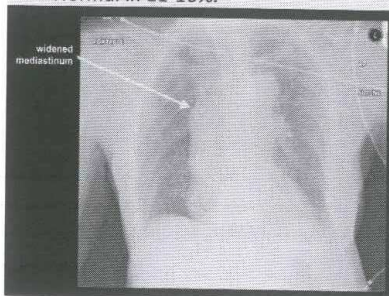
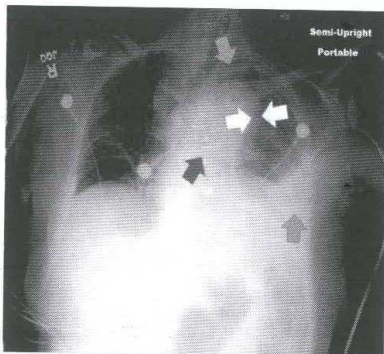
COMPLICATIONS

- Suspect if hypotensive (check for limb discrepancy!)
 - Aortic rupture
 - Aortic regurgitation
 - Acute Myocardial Infarction
 - Cardiac Tamponade
 - End-organ ischaemia – brain, limbs, spine, renal, gut, liver
 - Death

INVESTIGATIONS

- Bedside
 - ECG
 - Normal
 - Inferior ST elevation (right coronary dissection) but can be any STEMI (0.1% of STEMI are dissections)
 - Pericarditis changes, electrical alternans (tamponade)
- Laboratory
 - Leukocytosis/ Creatinine elevation with renal artery involvement
 - Troponin elevated if dissection causes myocardial ischaemia
 - D-dimer – if negative dissection is very unlikely, but not sufficient to rule out
 - X-match
 - Various biomarkers being investigated (e.g. elastin fragments, d-dimer, smooth muscle myosin heavy-chain protein)
- Imaging
 - CXR

- Widened mediastinum (56-63%), the **most reliable sign**
- Abnormal aortic contour (48%),
- Aortic knuckle: Double calcium sign >5mm (14%),
- Pleural effusion (L>R),
- Tracheal shift to the right,
- Left apical cap,
- Fractures of the first and second ribs
- Deviation of the nasogastric tube to the right
- Depressed left main stem bronchus
- Normal in 11-16%.



- **Echocardiography**
 - **Transthoracic (TTE)** 75% diagnostic Type A (ascending), 40% descending (Type B). Good for Aortic Regurgitation.
 - **Transoesophageal (TOE).** Much higher sensitivity / specificity, though operator-dependent, need sedation, and is less available. Useful in ICU / perioperative. Upper ascending aorta and arch not well visualised
- **Helical CT (CT MEDIASTINUM)**
 - Useful screen for widened mediastinum.
 - Newer multiplane/slice scanners may now negate additional need for TOE or aortography to plan operative management.
- **Aortography**
 - Was the traditional gold standard, delineating aortic incompetence and associated branch vessel involvement as well.
- **MRI / MRA**
 - Excellent sensitivity and specificity limited by availability.

ED MANAGEMENT OF AORTIC DISSECTION

- **Emergent priorities**
 - Control BP
 - Control bleeding
 - Fluid resuscitation
- O₂
- Wide bore IV access (Swan sheath)
- Invasive monitoring
- Warn blood bank (**X-match 6U** + need for other products)
- Correct coagulopathy
- Control HR and BP (**aim for P 60-80 and BP 100-120 SBP**)
- IV beta blocker (propranolol, esmolol or labetalol) combined with vasodilators (e.g. GTN, labetalol, SNP)
- Start β -blocker first to avoid increased aortic wall stress from reflex tachycardia
- Refer to cardiothoracic surgeon

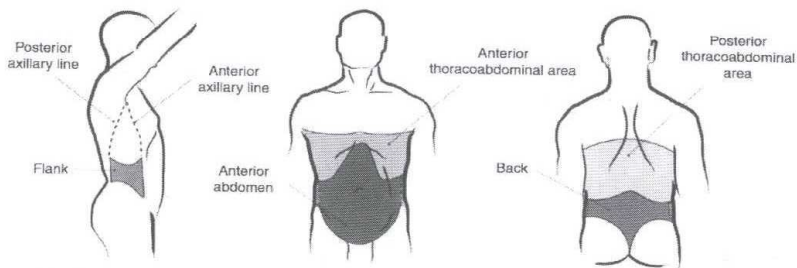
INDICATIONS FOR SURGERY

- *Persistent pain*
- *Type A*
- *Branch Occlusion*
- *Leak*
- *Continued extension despite optimal medical management*

CHAPTER 7. ABDOMINAL TRAUMA

OVERVIEW

- Assessment of abdominal trauma requires the identification of immediately life-threatening injuries on primary survey, and delayed life threats on secondary survey.
- Abdominal trauma is classified as **blunt or penetrating**, assessment and management is modified accordingly.
- **BLUNT ABDOMINAL INJURY**
 - Blunt abdominal injuries often managed conservatively, though interventional radiology and surgery are indicated for severe injuries.
 - Common mechanisms include road traffic crashes, falls, sports injuries and assaults
 - Organs most affected are: **Spleen > Liver > Small and Large Intestine.**
- **PENETRATING ABDOMINAL INJURY**
 - Most patients with significant penetrating injury require laparotomy; there are differences in the management of projectiles (e.g. gunshot wounds) and non-projectiles (e.g. stabbings)
 - Any wound between the **nipple line (T4) and the groin creases anteriorly**, and **from T4 to the curves of the iliac crests posteriorly** is potentially a penetrating abdominal injury. If the wound was caused by a projectile, then a penetrating abdominal injury could result from an entry wound in almost any part of the body.
- There are the 4 regions of the abdomen to consider in penetrating injury:
 - **Anterior abdomen**
 - Between the anterior axillary lines; bound by the costal margin superiorly and the groin crease distally.
 - **Thoracoabdominal area**
 - The area superiorly delimited by the fourth intercostal space (anterior), sixth intercostal space (lateral), and eighth intercostal space (posterior), and inferiorly delimited by the costal margin (definitions vary — a pragmatic approach is to use the nipple line as the upper boundary... in non-obese men at least!).
 - Injuries in the region increase the likelihood of chest, mediastinal, and diaphragmatic injuries.



- **Flanks**
 - From the inferior costal margin superiorly to the iliac crests; bound anteriorly by the anterior axillary line and posteriorly by the posterior axillary line.
- **Back**
 - Between the posterior axillary lines extending from the costal margin to the iliac crests.

I. BLUNT ABDOMINAL TRAUMA

ASSESSMENT

- **Primary survey**
 - Use a coordinated team-based systematic approach aimed at identifying, prioritising and treating immediate and delayed life-threats.
 - Abdominal and pelvic injuries may cause life-threatening haemorrhage.
 - Initial examination of the abdomen is best performed in the 'C' phase of the primary survey, with the mindset of '*Find the bleeding, stop the bleeding*' (x 16G cannula)
 - **Activation of massive transfusion protocol** if needed
- **Secondary survey** (search for signs that indicate need for emergency laparotomy)
 - **Inspection**
 - Abrasions, Bruising, **Lap belt** (30% chance of mesenteric or intestinal injury)
 - Retroperitoneal haemorrhage: ecchymosis of the periumbilical area (**cullen's sign**) and the flanks (**Grey-Turner's Sign**)
 - Genital and perineum
 - **Palpation**
 - Fullness: haemorrhage
 - Crepitation of lower rib cage: hepatic or splenic injury
 - Peritonism: ruptured viscus with leakage
 - **Rectal or Vaginal Examination**

INVESTIGATIONS

- **Trauma series** (e.g. C-spine X-Ray, CXR, Pelvis X-Ray)
- **Trauma blood panel** (e.g. FBC, U&E, LFTs, Lipase, Coags, Group and hold, BHCG)
- **Imaging** (bedside FAST scan, +/- CT abdomen if haemodynamically stable and imaging warranted)
- Insert gastric tube and IDC

IMAGING IN BLUNT ABDOMINAL TRAUMA

- In the absence of physical signs that indicate a need for immediate emergency laparotomy, imaging can be used to determine if emergency laparotomy is indicated, and help prioritise, identify and guide the management of other injuries:
 - **FAST**
 - **Diagnostic peritoneal lavage (DPL)** DPL is now rarely performed due to the advent of the FAST scan. Its main role is when FAST and CT are unavailable or in mass casualty situations.
 - **CT ABDO/PELVIS**

II. PENETRATING ABDOMINAL TRAUMA

ASSESSMENT

- In patients with penetrating abdominal injury, if immediate emergency laparotomy is not indicated then once the patient is stabilised we have to answer 2 questions that act as key decision nodes guiding our approach:
 - **Does the wound penetrate the peritoneum?**
 - **Is there intraperitoneal organ injury?**
- Only two-thirds of anterior abdominal stab wounds violate the peritoneum, and only half of these require operative intervention.

- **Assessment for peritoneal penetration:**
 - **Local Wound Exploration:** involvement of abdominal fascia is considered a positive result.
 - **CXR:** peritoneal penetration is confirmed by **free air under diaphragm**, but absence of free does not rule it out.
 - **Ultrasound:** peritoneal penetration is confirmed by **free fluid in the abdomen** or evidence of abdominal fascia violation, but absence of these findings does not rule it out.
 - **DPL:** this is invasive and not specific for injuries requiring operative intervention. It may be use if ultrasound is unavailable and some advocate it for thoracoabdominal wounds.
 - *A positive result is >100,000 RBCs/hpf (Red Blood Cells per High Power Field) for anterior abdominal wounds, and 10,000 RBCs/hpf for thoracoabdominal wounds that are at higher risk of diaphragmatic injury.*
 - Some suggest using the lower threshold for anterior wounds as well, but this leads to a higher negative laparotomy rate.
- If the fascia is intact (i.e. all of the above are confirmed negative, excluding peritoneal penetration):
 - *The wound can be cleaned and closed in the ED*
 - *If there are no other concerns the patient may be discharged*
- If local wound exploration is inadequate and abdominal fascia injury cannot be excluded, or there is evidence of peritoneal penetration, then further investigation is needed to assess for intraperitoneal injury.

IMAGING & INVESTIGATION OF PENETRATING ABDOMINAL INJURIES

- Similar to blunt abdominal trauma, a coordinated team-based systematic approach is used with the aim of identifying, prioritising and treating immediate and delayed life-threats
- **If emergency laparotomy is *not* indicated**, there are two options for identifying intraperitoneal injury:
 - *CT Abdomen*
 - *Direct laparoscopy*
- CT abdomen (97% sensitive for peritoneal violation) is usually performed to look for evidence of peritoneal penetration and intraperitoneal injury:
 - *Free air*
 - *Free fluid*
 - *Bowel wall thickening*
 - *Wound tracts adjacent to a hollow viscus solid organ injury*
- An alternative in some centers is direct laparoscopy, which is often performed for left thoracoabdominal wounds due to the risk of diaphragmatic injury (17%) and may allow repair.
- **If the peritoneum is penetrated**, then the options are:
 - **Laparotomy** if there is intraperitoneal injury requiring operative repair (e.g. Colon perforation), or
 - **Observation with serial examination +/- fast scans and serial full blood count** checks for 24 hours if no intraperitoneal injury (some injuries such as those the pancreas or a hollow viscus may not be detectable initially and may present later).
- The option of selective non-operative management of anterior abdominal wounds is made based on the type of intraperitoneal injury and the experience of the trauma center.

III. SPECIFIC ABDOMINAL INJURIES

1. GUNSHOT WOUNDS

- Assessment and management is modified compared to non-projectile penetrating abdominal trauma (e.g. stab wounds)
 - Abdominal gunshot wounds are more likely to penetrate the peritoneum (80%), and those that do are more likely to cause intraperitoneal injury (90%)
 - Bullets and similar missiles are higher velocity and may ricochet resulting in unpredictable wound tracts
- **DIFFERENCES IN ASSESSMENT:**
 - Local wound exploration should only be performed if the projectile was low velocity with a presumed tangential tract.
 - **CT abdomen with IV contrast** is the optimal method for determining both peritoneal penetration and intraperitoneal injury unless emergency laparotomy is indicated. It also identifies the missile pathway. Triple contrast is used if suspected gastrointestinal injury (**Triple-contrast CT** consists of giving oral, IV, and rectal contrast medium).
 - **DPL** is an alternative for detecting intraperitoneal injury if CT is not available, using a threshold of 5 to 10,000 RBCs/hpf for a positive result.
 - **Direct laparoscopy** is also useful for left thoracoabdominal gunshot wounds.
- **DIFFERENCES IN MANAGEMENT:**
 - Traditionally all gunshot wounds with peritoneal penetration undergo laparotomy
 - In centers experienced with the management of GSWs selective non-operative management may be used, such as isolated liver injuries (RUQ) and patients who remain hemodynamically stable with no evidence of peritonism.
- **MANAGEMENT**
 - **Resuscitate:** IV Fluid, Blood transfusion
 - **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
 - **Tetanus Prophylaxis**
 - **Nasogastric tube.**
 - **Urinary catheter**
 - **Keep nil by mouth.**
 - **Refer to surgical team.**

2. DUODENAL INJURY

- **ASSESSMENT**
 - Suspect in unrestrained drivers in frontal impact MVC and in patients who sustain direct blows to the abdomen, e.g. from bicycle handle bars.
 - Abdominal pain and tenderness
 - Bloody gastric aspirate
 - Retroperitoneal air on abdominal X-ray or CT abdomen
 - Can be confirmed by double contrast CT abdomen
- **MANAGEMENT**
 - **Resuscitate:** IV Fluid,
 - **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
 - **Keep nil by mouth, Refer to surgical team.**

3. SMALL INTESTINE INJURY

• ASSESSMENT

- Clinical signs can be minimal initially
- Usually a deceleration injury (e.g. MVC with lap belt)
- May involve bowel wall and / or mesenteric avulsion with subsequent intraperitoneal bleeding and devascularisation of bowel
- Coexistent lumbar distraction fracture (**Chance fracture**) may be present
- *An abdominal seatbelt sign mandates definitive imaging*
- May be missed on early FAST scan and CT abdomen — DPL or repeat examination may be required

• MANAGEMENT

- **Resuscitate:** IV Fluid,
- **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
- **NGT/ Keep nil by mouth.**
- **Refer to surgical team.**

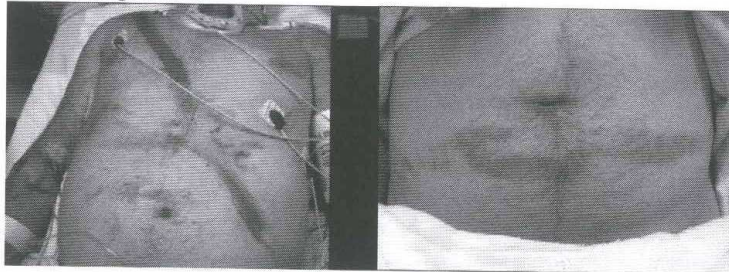


Figure 4.7.1. seat belt injury

4. PANCREATIC INJURY

• ASSESSMENT

- Classically due to a direct blow, e.g. motorbike handlebars
- Abdominal pain +/- vomiting
- **Double contrast CT abdomen and amylase / lipase** may initially be normal

• MANAGEMENT

- Usually conservative, rarely surgical exploration and repair are needed

5. DIAPHRAGM INJURY

• OVERVIEW

- Diaphragmatic injury can be a challenging diagnosis and is missed on imaging about 50% of the time. Comprises 0.8 to 8% of all closed blunt trauma and penetrating trauma case combined. More common in penetrating trauma — suspect if wound tract may extend between T4 and T12 levels
- Suspect also in severe blunt trauma (e.g. abdominal crush injury, ejection) associated with **respiratory distress if left-sided** and **deep visceral pain if right-sided**.
- Most commonly posterolateral left hemidiaphragm in blunt trauma, as liver diffuses force and protects the right diaphragm.
- Herniated organs: **stomach > small and large bowel > spleen > liver**
- Mortality 14 to 50% — associated abdominal injury is very common

- **ASSESSMENT**

- **History**

- Trauma/ Frequently no symptoms
 - If delayed: SOB, post prandial epigastric pain, thoracic pain
 - Rarely gastric herniation or volvulus (vomiting, sepsis if strangulation has occurred)
 - Shoulder pain

- **Examination**

- Frequently no signs, Tachypnoea, Tachycardia, hypotension (strangulation)
 - Decreased Spo₂, Fever, Decreased chest expansion on affected side (left > right x 3)
 - Dullness to percussion
 - Decreased air entry at affected base
 - Bowel sounds in the chest
 - Schaphoid abdomen
 - Abdominal tenderness, peritonism

- **INVESTIGATIONS**

- **Chest x-ray** may be normal (~50%) or show:

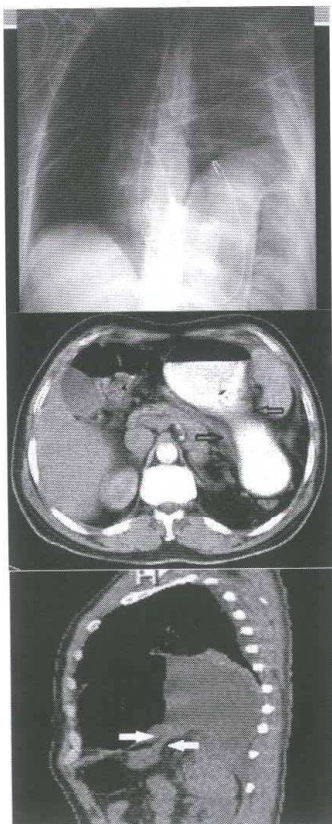
- *Elevation or "blurring" of the hemidiaphragm*
 - *Hemothorax*
 - *An abnormal gas shadow that obscures the hemidiaphragm*
 - *The gastric tube being positioned in the chest*

- **CT;** in some centers sensitivity is as high as 95% for MDCT (machine and radiologist dependent):

- **The Collar sign** (or **hour glass sign**) = a waist-like constriction of the herniating hollow viscus at the site of the diaphragmatic tear.
 - **The Dependent viscera sign** = viscera are unsupported posteriorly by the injured diaphragm and fall to a dependent position against the posterior ribs.
 - Segmental non-recognition of diaphragm.
 - Focal diaphragmatic thickening.
 - Thoracic fluid abutting the abdominal viscera.
 - Associated abdominal injuries.

- **Laparoscopy** (surgical 'eye-ogram')

- Gold standard (along with laparotomy)
 - Often performed if penetrating wound in left thoracoabdominal area.



MANAGEMENT

- ATLS approach
- **Resuscitate:** IV Fluid, **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
- Treat associated injuries
- Decompress stomach with a gastric tube/ **Keep nil by mouth.**
- **Refer to surgical team.**

6. LIVER TRAUMA

OVERVIEW

- Liver trauma may result from blunt or penetrating abdominal injury
- The liver is the most commonly injured organ in penetrating abdominal trauma

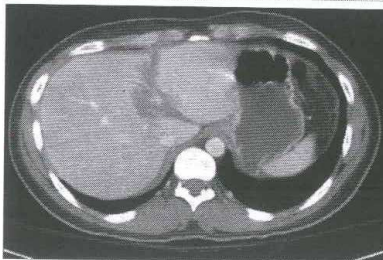
ASSESSMENT

- Abdominal pain, localized tenderness (RUQ)
- Possible hemorrhagic shock
- **CT abdomen with IV contrast is the investigation of choice** (liver injuries are graded I to VI according to severity)

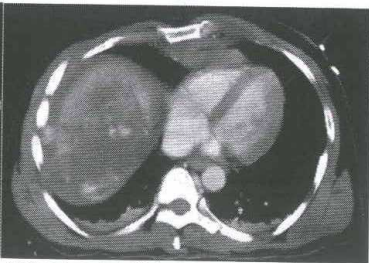
GRADING

- American Association for Surgery of Trauma Organ Injury Scale based on:

GRADE	DESCRIPTION OF INJURY
I	Small (<10%, < 1cm)
II	Moderate (10-50%, < 10cm)
III	Large (>50%, > 10cm or expanding)
IV	Large with parenchymal disruption (>25% of a hepatic lobe)
V	Large with parenchymal disruption (> 75% of a hepatic lobe) or juxtahepatic venous injury
VI	Hepatic avulsion



Axial post-contrast CT shows capsular tear, stellate hepatic laceration with injury close to the hilum, no hematoma, grade III



Axial post-contrast CT shows capsular tear, large stellate hepatic laceration involving segments 7 and 8, active hemorrhage, and subcapsular hematoma, grade IV

MANAGEMENT

- ATLS approach
- **Resuscitate:** IV Fluid,
- **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
- **Keep nil by mouth.**
- **Refer to surgical team:**
 - Most hemodynamically stable injuries can be managed non-operatively
 - **Angiography with embolization** should be considered.

7. SPLEEN TRAUMA**• OVERVIEW**

- Splenic trauma may result from blunt or penetrating abdominal injury
- The spleen is the most commonly injured organ in blunt abdominal trauma

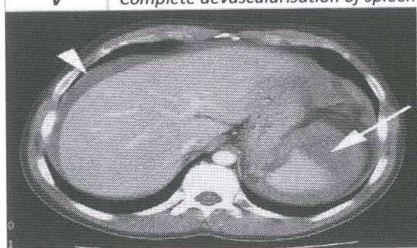
• ASSESSMENT

- Abdominal pain, localized tenderness (LUQ)
- Possible hemorrhagic shock
- **CT abdomen with IV contrast is the investigation of choice** (spleen injuries are graded I to V according to severity)

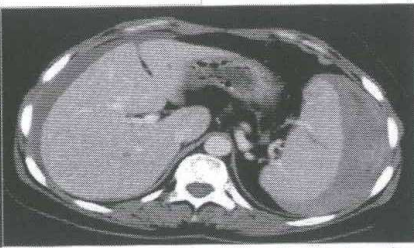
• GRADING

- American Association for Surgery of Trauma Organ Injury Scale based on:

GRADE	DESCRIPTION OF INJURY
I	Small (<10%, < 1cm)
II	Moderate (10-50%, < 5cm)
III	Large (>50%, > 5cm or expanding)
IV	Large with partial devascularisation (>25%)
V	Complete devascularisation of spleen



Rupture of the spleen. This CT shows Rupture of the anterior half of the spleen caused by blunt trauma in falling from a horse. Haemorrhage is seen within the splenic bed (arrow) along with free blood around the liver



Contrast-enhanced CT scan : sub capsular splenic hematoma that involves more than 50% of surface area

• MANAGEMENT

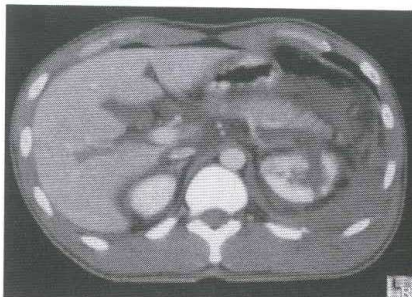
- ATLS approach
- **Resuscitate:** IV Fluid,
- **Analgesia: Morphine IV titrated to effect:** Analgesia should never be withheld until the patient has seen the surgeon.
- **Keep nil by mouth.**
- **Refer to surgical team:**
 - Most hemodynamically stable injuries can be managed non-operatively (especially Grades I to III)
 - Injuries involving the hilum or avulsion often require surgery (Grade IV or V)
 - Angiography with embolization should be considered.
 - Patients with functional asplenism will need immunisations and follow up similar to post-splenectomy patients

8. KIDNEY TRAUMA**• OVERVIEW**

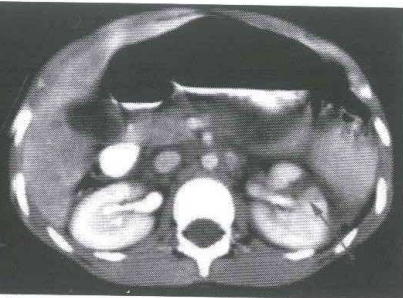
- Most genitourinary injuries are not immediately life-threatening.
- Renal pedicle injury can lead to life-threatening hemorrhage and renal ischemia.

• ASSESSMENT

- Clinically significant injuries will have at least one of:
 - **Macroscopic haematuria** (About 5% of renal injuries and up to 20% of renovascular injuries lack even microscopic hematuria)
 - Loin tenderness and/ or swelling
 - Haemodynamic instability
 - Fracture of the lower posterior ribs, lower thoracic or lumbar vertebrae may also be present.



Contrast-enhanced CT of the mid-abdomen reveals a linear zone of low attenuation through the left kidney with surrounding perinephric standing characteristic of a deeper renal laceration.



Grade I renal laceration displaying the extension of injury to the vascular pedicle.

• IMAGING

- **CT abdomen with IV contrast is the investigation of choice** (severity is graded I to V)
- **IVP** (intravenous pyelogram) is an option if CT is unavailable or imaging needs to be carried out in the operating theatre, but is less sensitive and does not visualize non-urologic injuries
- **Renal angiography** is rarely required

• GRADING

- American Association for Surgery of Trauma Organ Injury Scale based on:

GRADE	DESCRIPTION OF INJURY
I	haematuria, no parenchymal involvement, subcapsular, normal urogram
II	non-expanding, confined to retroperitoneum, < 1cm, no urinary extravasation
III	>1cm involving renal cortex (no urinary extravasation or collecting system involvement)
IV	cortex, medullary and collecting system or vascular involvement
V	shattered or an avulsed kidney

MANAGEMENT

- ATLS approach
- Urology consult
- Most renal injuries (Grades I to III, and most Grade IV injuries) can be managed **conservatively**, as they tend to heal spontaneously.
- Surgical repair is needed for urinary extravasation or if ongoing bleeding or hemodynamic instability due to renal injury.
- Alternatives to operative repair are interventional radiology to embolise bleeding vessels or to stent dissected renal arteries, and urinary extravasation may be amenable to stenting. Grade V injuries (avulsed kidneys) need operative intervention and often require nephrectomy

9. HAEMATURIA IN TRAUMA**• OVERVIEW**

- Haematuria in trauma may be microscopic (with or without symptoms) or macroscopic
- In general, the greater the degree of hematuria the greater the risk of significant intra-abdominal injury (including non-urinary tract structures). About 5% of renal injuries and up to 20% of renovascular injuries lack hematuria — even severe injuries such as renal artery injury or ureteropelvic disruption may present without hematuria.

• GOALS

- *Find source of bleeding along urological tract*
- *Treat cause*
- *Support blood volume*
- *Identify associated injuries*

ASSESSMENT**1. MICROSCOPIC HAEMATURIA IN BLUNT TRAUMA**

- If the patient is asymptomatic the yield of injuries requiring intervention in this setting is extremely low:
 - No further imaging is needed
 - Arrange repeat urinalysis (e.g. in a week's time) and close follow up by a GP
 - Some experts advocate imaging in pediatric patients with asymptomatic hematuria following blunt abdominal trauma as they are more vulnerable to significant renal injury; cut off values vary, with values from 5 to 50 RBCs/hpf being suggested.
- Hematuria with <5 RBCs/hpf can be caused by urinary catheter insertion
- If the patient is significantly **symptomatic**, they may have associated non-urinary intrabdominal or retroperitoneal injury: **CT abdomen with IV contrast.**

2. MACROSCOPIC HAEMATURIA IN BLUNT TRAUMA

- 50% of such patients have renal injuries, and a further 15% have injuries to other intra-abdominal organs
 - **CT abdomen with IV contrast and CT cystogram**

3. HAEMATURIA IN PENETRATING TRAUMA

- Surgical exploration +/- CT if stable
- Perform urethrogram if suspected urethral injury
- Displaced fractures of the pelvic ring, particularly **'butterfly fractures'**
- Gross hematuria, difficulty placing a urinary catheter
- Classic clinical features are uncommon:
 - *Blood at the meatus*
 - *Perineal / scrotal haematoma*
 - *High riding prostate on examination*
- Skeletal injuries associated with genitourinary injury:
 - **Pelvic fractures**
 - Posterior urethral injury (above the urogenital diaphragm) and bladder injury
 - **Perineal straddle injury**
 - Anterior urethral injury
 - **Fracture of the lower posterior ribs, lower thoracic or lumbar vertebrae**
 - Renal or ureteral injuries
- **MANAGEMENT**
 - ATLS approach and Urology referral if significant identifiable injury.

CHAPTER 8. FRACTURES

I. CERVICAL SPINES FRACTURES

1. HYPERFLEXION

- o Causes compression of the anterior aspects of the vertebral bodies and posterior ligament complex distraction
- o This leads to:
 - **Chance fractures:** horizontal fracture through body, pedicle and posterior elements of the vertebrae
 - **Tear drop fractures:** fracture of the anteroinferior vertebral body (**teardrop sign**)
 - **Rupture of posterior ligament.**
 - **The odontoid peg may also be fractured:** by sudden severe flexion.



Figure 4.8.1. Chance fracture



Figure 4.8.2. Tear Drop Fracture

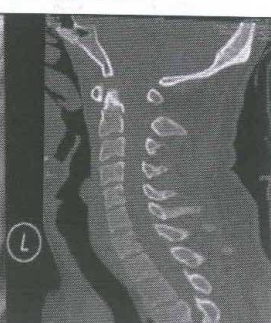
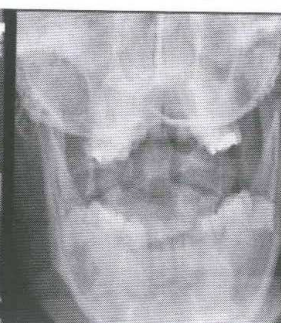
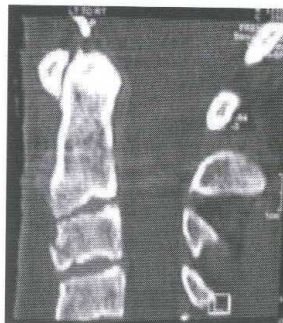
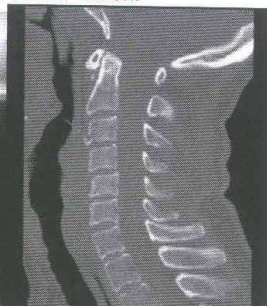


Figure 4.8.3. Odontoid Peg Fracture

2. FLEXION AND ROTATION

- o 50%-80% of cervical spine injuries and most thoracolumbar are caused by this mechanism.
- o This mechanism causes disruption of the posterior ligament complex and the posterior column.
- o The facet joints, lamina, transverse processes, and vertebral bodies may fracture
- o Relatively flat facet joints may dislocate without fracture
- o Spinous processes of C6/7 can also be avulsed by interspinous ligaments the so-called **clay-shovelers fracture**.

- o All **intervertebral ligaments may tear** and the upper vertebral body can be displaced relative to the one below

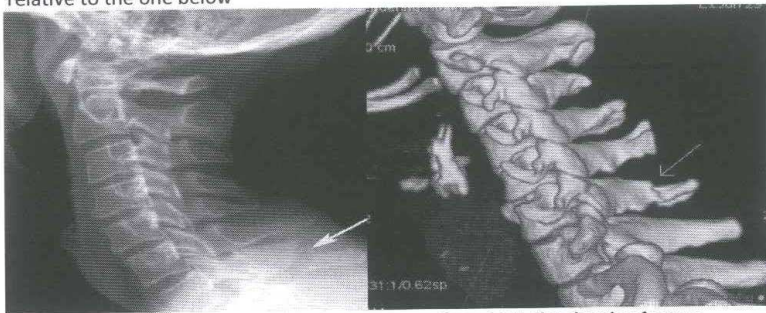


Figure 4.8.4. Clay-shovelers fracture

3. HYPEREXTENSION

- o Damages the anterior column associated with anterior fracture of the anterior inferior aspect of vertebral body.
- o The posterior aspect of the vertebral body may be crushed with a risk of retropulsion of bony fragments or the intervertebral disc into the spinal canal



- o **Hangmans fracture:** fracture through pedicles of C2 following hyperextension with distraction or compression.
- o Results from judicial hanging (rather than suicidal which causes asphyxiation) or from striking chin on steering wheel in a collision

4. ROTATION

- o Rarely occurs in isolation
- o Primary injury occurs to the posterior ligament complexes, and is often unstable
- o May result in **facet joint dislocation**.

5. COMPRESSION

- o This mechanism is common in thoracic and lumbar spine injuries and results in wedge fractures.
- o **The Jefferson Fracture of C1:** is a specific cervical spine fracture caused by an axial loading mechanism (eg. a weight landing on patient's head, or patient landing on their head after a fall). The characteristic features are:

- The atlas is compressed between the occipital condyle and C2
- The laminae and pedicles are fractured and transverse ligament holding peg in position can be torn.
- The skull and C1 may slide forward on C2.
- There can be significant shift before compression of spinal cord occurs as 1/3rd of the space of the spinal canal is occupied by the odontoid peg, 1/3rd by areolar tissue and 1/3rd by the spinal cord itself.

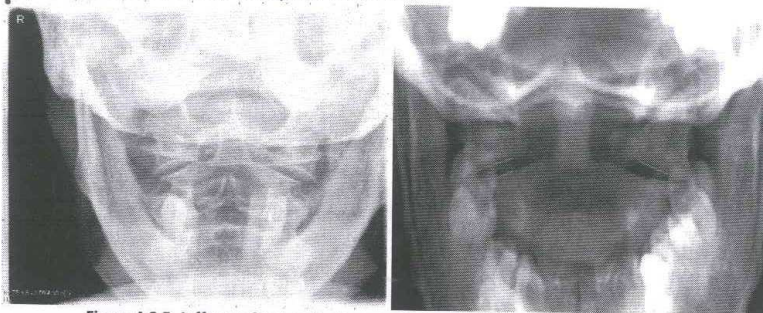


Figure 4.8.5. Jefferson fracture (note overriding of the lateral masses of C1 on C2)



Figure 4.8.6. CT Images of Jefferson Fracture (Different patients scan)

DIFFERENCES BETWEEN ADULTS AND CHILDREN

- The anatomy and relationships of the child's cervical spine is different to that in adults:
 - Children have relatively larger heads
 - Their ligaments and joint capsules are more lax
 - Their facet joints are more horizontal
 - Their vertebral bodies are wedge shaped
- This has the following consequences:
 - **Pseudosubluxation**: refers to the appearance of forward slippage of one vertebral body on another;
 - Pseudosubluxation of C2 on C3 occurs in 24% of under 8 year olds and of C3 on C4 in 14% of under 8 year olds.



Figure 4.8.7. Pseudosubluxation (note the apparent forward slippage of C2 on C3)

- o **SCIWORA** (Spinal Cord Injury without radiological abnormality) which is defined as objective signs of myelopathy as a result of trauma with no evidence of fracture or ligamentous instability on plain radiographs or tomography.
- o It is more common in children and is reported to occur in up to 30% of spinal cord injuries in children. Younger children below 10 yrs are also more likely than older children to:
 - Injure the upper cervical spine (C1-C4) compared with lower c-spine
 - Dislocate the cervical spine
 - Injure the spinal cord itself
 - The cervical spine takes on its adult form from about the age of 8 years.

CLINICAL ASSESSMENT

- For the purposes of clearing the cervical spine, patients can be divided into two groups:
 - o Conscious and cooperative
 - o Unconscious and/or uncooperative

1. Conscious cooperative patients

- This is the most commonly encountered group of patients who present to the ED or pre-hospital practitioner.
- They have a low incidence (less than 3%) of cervical spine injury and are able to cooperate with clinical assessment.
- Therefore, a focussed history and examination can be used to clinically clear their necks – various clinical decision rules have been developed to be used in these patients:
 - o *Nexus Low Risk Criteria*
 - o *Canadian Cervical Spine Rules*

2. UNCONSCIOUS / UNCOOPERATIVE PATIENTS

- These patients are not able to have their cervical spines cleared clinically as a reliable clinical assessment cannot be made.
- These patients require imaging to clear their spines.

NEXUS LOW RISK CRITERIA (NLC)

- This was developed from a prospective study of patients undergoing cervical spine radiography in 21 centres in the USA.
- The study looked at 5 criteria; if all were negative the patient was classified as having a low risk of injury:

NEXUS LOW PROBABILITY CRITERIA

- o *No midline cervical tenderness*
- o *No focal neurological deficit*
- o *Normal alertness*
- o *No intoxication*
- o *No painful distracting injury*

CANADIAN C-SPINE RULE (CCR)

- The decision rule resulting from this study asks 3 questions:
 - o Is there any high-risk factor present which mandates radiography? *Age ≥ 65 or dangerous mechanism of injury or paresthesia of extremities?*
Yes>>> Radiography
 - o Is there any low-risk factor present that allows safe assessment of the range of neck motion? *Simple rear MVC or sitting position in ED or ambulatory anytime or*

Delayed onset of pain or Absence of midline tenderness?

No>>> Radiography

- o Is the patient able to actively rotate their neck 45° to the left and right?

Unable>>> Radiography

- Unlike the NEXUS rule, this study excluded children <16 years of age, and all patients with a Glasgow Coma Scale (GCS) score of <15.
- **Children:** There is no robust evidence base for a clinical rule-out for cervical spine injury in children less than 10 years of age.

INVESTIGATION STRATEGIES

- Patients, whose cervical spine cannot be clinically cleared, will require imaging to permit identification or exclusion of significant injury.

a. Plain cervical spine series

- Plain radiographs are not adequate to exclude significant cervical spine injury in unconscious patients and these patients will require CT (or MRI) imaging.
- Normal imaging of the cervical spine consists of three views: **The Lateral, Antero-posterior (AP) and odontoid peg views.**
- In children under 5, the PEG view is considered unnecessary.

b. Swimmers view:

- In the case of an inadequate lateral view:
 - o An arm pull view in which the patients arms are pulled down to try to lower the shoulders so the lower cervical spine can be visualised or
 - o A swimmer's view is obtained in which the arm nearest the x-ray machine is elevated and the arm nearest the plate is kept extended. This view can be difficult to interpret due to overlying bones.

c. Flexion / extension views

- *There is no role for flexion/extension views in the acutely injured neck.*

d. Computerised Tomography (CT)

- The indication for CT scanning needs to be carefully considered because patients undergoing CT of their whole cervical spine have a **14-fold increase in the dose of radiation to their thyroid gland** compared with standard three view plain radiography.
- The recent introduction of spiral CT has reduced radiation dose and is faster, with a reported sensitivity as high as 99% and a specificity of over 93%.

e. Magnetic Resonance Imaging (MRI)

- MRI scanning is very sensitive for soft tissue injuries including ligament injuries, disc herniation and haemorrhage, which are less well visualised on CT. Many of these injuries will not be clinically significant, but a minority will represent unstable injuries.
- MRI is, however, less sensitive than CT at imaging the posterior elements of the spine and the craniocervical junction. MRI is indicated if there is any neurology referable from the cervical spine, or if there is severe pain, despite a normal CT scan as some unstable ligamentous injuries may only be seen on MRI
- **Current NICE Guidelines do not recommend the routine use of MRI scanning to clear the cervical spine.**
- The current NICE recommendation for imaging the cervical spine of a conscious co-operative patient is to use 3 view plain radiographs, unless one of the following is present, in which case CT should be the primary modality of imaging (See **NICE CRITERIA FOR PERFORMING A CT CERVICAL SPINE SCAN/** Head injury section above).

II. FACIAL BONES INJURY

INTRODUCTION

- Although facial injuries are one of the most common problems seen in the ED, interpretation of facial x rays remains a frequent cause of diagnostic error.

DEFINITION

- Injuries to the zygoma and the surrounding facial bones are confusingly referred to by a number of different terms including:
 - Malar fracture
 - Tripod fracture
 - Zygomatic complex fracture
 - Zygomaticomaxillary complex fracture
- All these terms refer to injuries of the zygoma with, in most cases, involvement of the maxilla or temporal bone. The sutures connecting the zygoma to adjacent bones may also be disrupted. Whilst it is important to delineate the individual components of a facial injury, in most instances this can only be done accurately by CT scan at a later stage.
- For clinical clarity in the ED, it is therefore suggested that the majority of injuries involving the zygoma and surrounding bones are either referred to as:
 - **Zygomatic arch fractures** i.e. Fractures predominantly affecting the zygomatic arch
 - **Zygomaticomaxillary complex (ZMC) injuries** i.e. Those fractures mainly involving the zygoma, maxilla and / or the orbital rim.

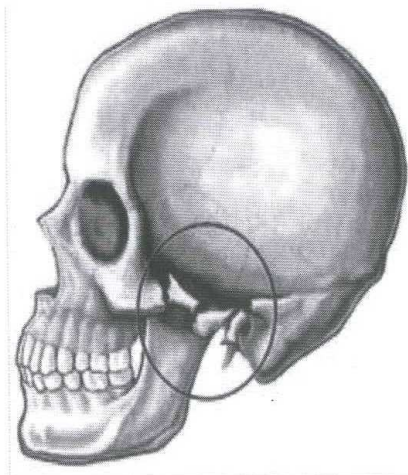


Figure 4.8.8. Zygomatic arch fracture

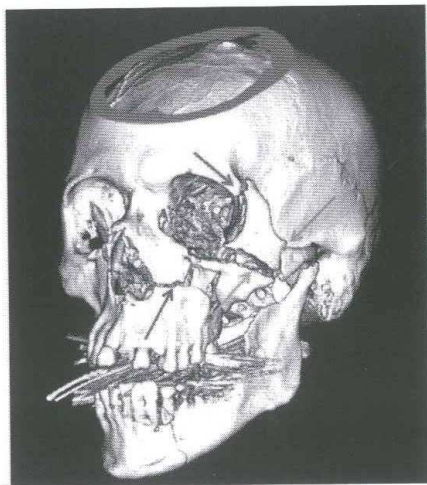


Figure 4.8.9. Zygomaticomaxillary complex fracture

- These descriptions do not encompass other important midface fractures which may be differentiated clinically in the ED:
 - **Nasal and Nasoethmoidal fractures**
 - **Orbital floor fractures**
 - **Le Fort type fractures**

CLINICAL ASSESSMENT

- The initial assessment of a patient with a facial injury must start with an ABCDE approach.
- In facial injuries, associated head and neck injury must also be considered.

History

- When taking a history, the EP must include document the mechanism and circumstances of the injury (e.g. interpersonal violence, road traffic accident) as well as the presence or absence of head injury symptoms such as loss of consciousness, amnesia and vomiting.
- For injuries of the **ZMC** and **zygomatic arch**, the history must also include;
 - **Visual disturbance** indicating possible orbital or globe injury
 - **Alteration in bite or difficulty moving the jaw** suggests mandible, maxilla or zygomatic arch injury
 - **Sensory disturbance to the cheek and upper gum** a sign of infraorbital nerve injury
- For **nasal injury**, the history must include:
 - Previous nasal injury / deformity often a perceived nasal deformity is pre-existing.
 - Epistaxis this may be extensive with nasal trauma but a history of epistaxis alone is not predictive of a new nasal deformity.
 - Anticoagulant medication may complicate the management of post-traumatic epistaxis
 - Any persistent nasal discharge since the injury this symptom may indicate a nasoethmoid injury with CSF leak.
- The EP must also be aware of the association between facial injury and the abuse of children, women and vulnerable adults.
- If doubt exists, for example a delayed presentation or mismatch between the history and examination findings, then seek advice from a senior EM or paediatric clinician.

Examination

- A general examination is important to identify any potential threat to the airway and the systemic effects of bleeding. If airway compromise is identified or threatened, senior EM and anaesthetic support should be called urgently.
- **Face**
 - Using a look, feel and move approach:
 - **Look:** Look for the following, remembering to use a head light, nasal speculum and suction when examining the nose:
 - **Areas of swelling, bruising and bleeding:** persisting bleeding and / or discharge from the nose may indicate a nasoethmoidal fracture.
 - **Deformity of the nose and zygomatic arch depression** of the zygomatic arch (flattened face) is best identified by looking from above or below.
 - **Septal haematoma of the nose:** a haematoma on the side of the nasal septum which needs draining urgently to prevent a "**Saddle Nose Deformity**" from ischaemic necrosis.
 - **Evidence of injury to the eye(s):** the position of the eye and visual acuity should be checked. Enophthalmos and proptosis both indicate a significant orbital injury.
 - **Subconjunctival haemorrhage:** if the posterior limit of the haemorrhage cannot be seen, it is likely that blood has tracked round the eye from a fracture of the orbital wall. A clear posterior border suggests a direct blunt injury to the globe.
 - **Feel:** palpation of facial bony landmarks and an assessment of neurological function should be undertaken, specifically identifying:
 - **Zygomatic arch:** check for a step or flattening caused by a depressed fracture.

- **Periorbital region:** for the crepitus of surgical emphysema this indicates a fracture involving a sinus, usually the maxillary.
- **Intraorally:** to assess the maxilla in the upper buccal sulcus for tenderness or a step.
- Assessing **sensation to the skin** supplied by the infraorbital nerve
- **Move:** Movement of the eye and jaw must be assessed;
 - **Eye movement:** particularly upward gaze, may be restricted in orbital blow-out fracture *due to trapping of the herniated inferior rectus muscle*.
 - **Limited jaw movement:** caused by restricted movement of the coronoid process of the mandible under the zygomatic arch may be found in depressed fractures of the zygomatic arch.

INVESTIGATION STRATEGIES

- **Facial X rays**
 - X rays are the cornerstone of investigation of ZMC and zygomatic arch injury in the ED.
 - Having made the decision to order facial imaging, the clinician is then faced with a choice of different views to visualise the ZMC and zygomatic arch including:
 - **Lateral facial view**
 - **Occipitontental 15° (OM15) and occipitontental 30° (OM30) views**
 - **Submentovertical view**



Figure 4.8.10. Normal OM15 view of the face Figure 4.8.11. Normal OM30 view of the face

- Two facial views – the OM15 and OM30 – provide the best combination of accuracy in identifying midfacial fractures whilst minimising radiation exposure.
- In patients where clinical findings suggest a zygomatic arch fracture, a specific arch view such as the submentovertical, will facilitate identification of the fracture.
- **Other Radiological Investigations**
 - **CT Scan:** its use in the ED is restricted to either a second line investigation, usually initiated by the maxillofacial team, or when other injuries (e.g. cervical spine injury) prevent routine facial x-rays being performed.
 - The diagnosis of **orbital blow-out fracture** may be made on routine facial x rays (e.g. a **teardrop sign**) but CT scan remains the gold standard if this injury is suspected or identified.
 - **Focused Ocular Ultrasound (FOUS)** has been evaluated in the ED and found to be highly accurate in both diagnosing and excluding both orbital and ocular trauma.

- There can be no justification for ordering x-rays of the nasal bones for a patient with suspected nasal fracture.



TEAR DROP SIGN: There is a fracture of the inferior floor of the right orbit, and there is evidence of orbital contents (such as the inferior rectus muscle) bulging into the right maxillary sinus (yellow arrow). There is an air-fluid level in the sinus in this example (orange arrow), which is due to haemorrhage and is a very helpful radiographic sign when the fracture itself is less obvious.

CT is usually performed in these cases; in this example, it confirms the displacement of some orbital fat through the fracture (arrow).

BIOCHEMICAL INVESTIGATIONS

- In patients with nasal injury and persisting discharge from the nose, it can be difficult to differentiate between nasal secretion and CSF arising from a **nasoethmoidal fracture**.
- Although often advocated:
 - **Testing for the Presence of glucose**, which is present in CSF but not normally in nasal secretion, may be falsely positive due to contamination of nasal secretions by blood or tears.
 - **Halo sign or Ring sign:** Dab some of the blood on a tissue. If there is CSF mixed with the blood, it will move by capillary action further away from the centre than the blood will.

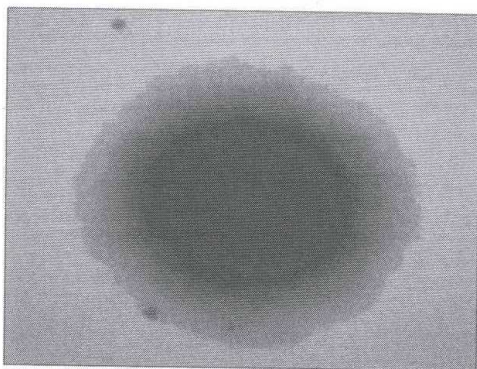
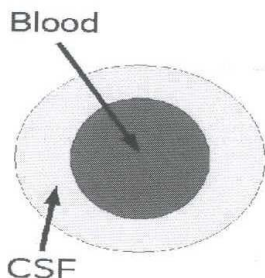
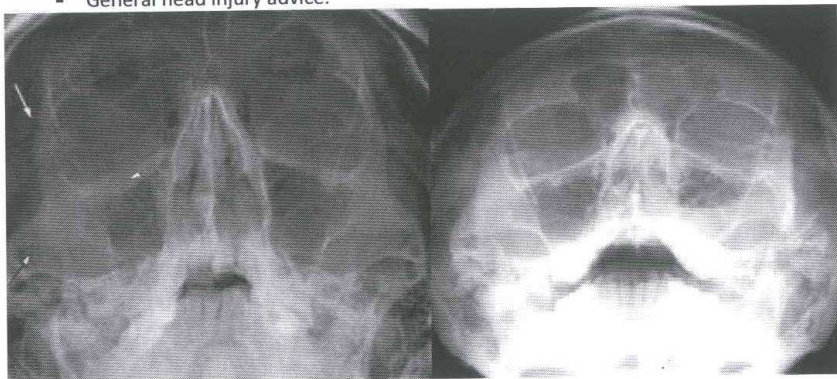


Figure 4.8.12. Halo sign

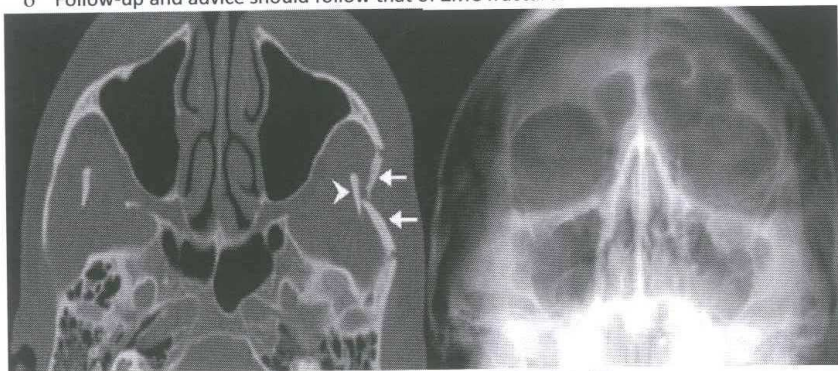
- **Beta-2 transferrin** (also known as the **Tau protein**) is almost exclusively found in the CSF and is a highly sensitive and specific test for the presence of CSF.
- *The presence of beta-2 transferrin in nasal discharge is the most accurate diagnostic test to confirm CSF rhinorrhea.*

1. ZMC FRACTURE

- o The vast majority can be managed expectantly until local swelling subsides and review by a maxillofacial surgeon at a time guided by local policy.
- o **If eye involvement** (e.g. reduced visual acuity or diplopia): urgent referral to a maxillofacial surgeon and / or an ophthalmologist.
- o **If infraorbital nerve involvement:** is not an indication for urgent referral.
- o Patients should be given **general advice** regarding their injury including:
 - Avoidance of nose blowing as this **may produce surgical emphysema**.
 - Not to occlude the nose when sneezing.
 - Application of ice packs to the area to reduce swelling.
 - Take regular analgesia.
 - General head injury advice.

**2. ZYGOMATIC ARCH FRACTURE**

- o As for ZMC fractures the majority of zygomatic arch fractures do not need urgent surgical intervention.
- o If there is restriction of mouth opening due to **trapping of the temporalis muscle or mandibular condyle**, is an indication for urgent referral to a maxillofacial surgeon.
- o Follow-up and advice should follow that of ZMC fracture.



Left sided orbital floor fracture with teardrop sign

3. ORBITAL BLOW-OUT FRACTURE

- o *Visual disturbance, limitation of eye movements and a teardrop sign on facial x ray are all signs of an orbital blow-out fracture: Immediate referral to an ophthalmologist or maxillofacial surgeon is essential.*
- o Facial CT will be needed to visualise the fracture in detail and plan surgical repair.
- o There is no evidence to support routine antibiotic prophylaxis in orbital floor fracture.

CLINICAL PRESENTATION

- Orbital blow-out fractures are usually the result of a direct blow to the orbit.
- This results in a sudden increase in the intraorbital pressure which in turn causes decompression by fracture of one or more of the bounding walls of the orbit.
- The trauma is usually substantial, but presentation and diagnosis may sometimes be delayed.
- This delay is usually due to intact orbital rim (by definition) and swelling making assessment of diplopia and extra-ocular movement difficult.
- Associated clinical findings of facial bones injuries may include:
 - o *Enophthalmos and proptosis: due to increased orbital volume*
 - o *Diplopia: due to trapping of the herniated inferior rectus muscle*
 - o *Orbital emphysema: especially when fracture is into an adjacent paranasal sinus*
 - o *Sensory disturbance to the cheek and upper gum: a sign of infraorbital nerve injury*
 - o *Restriction of mouth opening in ZMC Fracture: due to trapping of the temporalis muscle or mandibular condyle.*

4. NASAL FRACTURE

- o **Nasal fracture** is a clinical diagnosis and there is no evidence that immediate reduction of a displaced fracture practiced in some centres, is any better than delayed assessment and reduction.
- o However, displaced fractures must be reduced before 14 days as attempted closed reduction beyond this time may be impossible.
- o **Septal haematoma** is a rare problem but is more common in children due to the relative lack of bone in the nose which is softer and therefore more easily deformed. If identified, *a patient with a septal haematoma must be referred urgently to ENT for drainage and nasal packing.*
- o **Traumatic epistaxis** is common in nasal fracture and although occasionally severe, is usually self-limiting. It can be managed in a similar way to a non-traumatic nosebleed.
- o **If CSF rhinorrhoea** is confirmed, the patient should be **referred immediately** to an otolaryngologist for further assessment.

Uncontrolled epistaxis, CSF rhinorrhoea and septal haematoma are all indications for urgent ENT referral in nasal injury.

- The management of nasal injuries varies greatly across the United Kingdom and therefore a suggested flow chart is as follows:

NASAL INJURY**Full assessment including:**

- Cosmetic appearance
- Swelling
- Bleeding/ discharge
- Septal position and appearance

Are there any significant complications of nasal injury present?

NO

Yes

Obvious new deformity or new septal deviation

NO

Yes

Discharge home:

Ensure arrangement in place for ENT review at 7-10 days if:

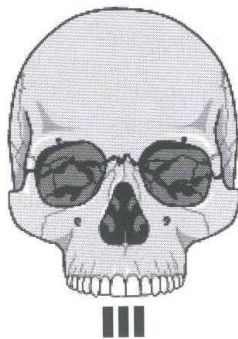
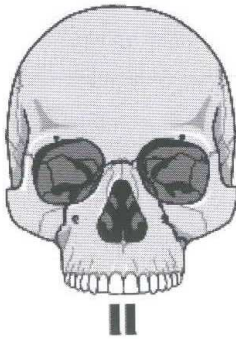
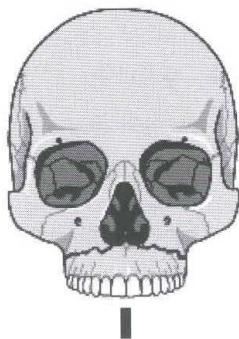
- Patient remains unhappy with cosmetic appearance once swelling has settled.
- Unable to breathe normally through each nostril

Complications include:

- Uncontrolled haemorrhage
- Septal haematoma/ abscess
- CSF rhinorrhoea

Refer ENT within 7-10 days of injury

Immediate ENT referral

5. LE FORT FRACTURES (MIDDLE THIRD)

- The Maxilla is a complex bone made up of strong buttresses but with areas of weakness around the maxillary sinus.

Le Fort I

- o Transverse fracture through floor of maxillary sinuses (only palate moves)

Le Fort II

- o Through nose, lower orbits and maxillary sinuses (pyramidal shaped fracture)

Le Fort III

- o Through orbits (craniofacial dysjunction) (separates the entire midface from the base of the skull)
- o Combinations occur and fractures are often comminuted

EXAMINATION

• Inspection

- Displacement: lengthening of the midface, bruising, lacerations
- Subconjunctival haemorrhage, enophthalmos, diplopia
- Bleeding/CSF from nostrils, or post-nasally
- Disruption of occlusion or dental arch
- Missing or loose teeth- bruising in centre of palate or buccal sulcus

• Palpation

- Rock maxilla against stable point e.g. upper basal skeleton
- Check orbital margins for palpable steps
- Check infra-orbital nerve

IMAGING

• Plain radiographs

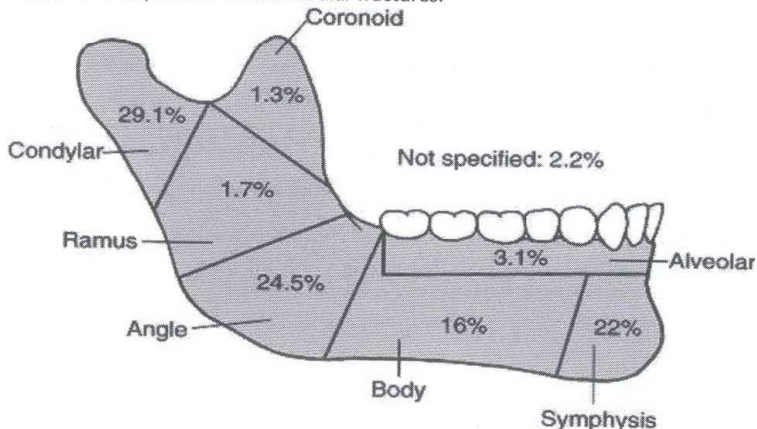
- Plain films are limited by their ability to penetrate through extensive soft tissue edema and to help distinguish between multiple planes of complex bony framework.
- OM15 and OM30 views
- Lateral view facial bones

• CT scans

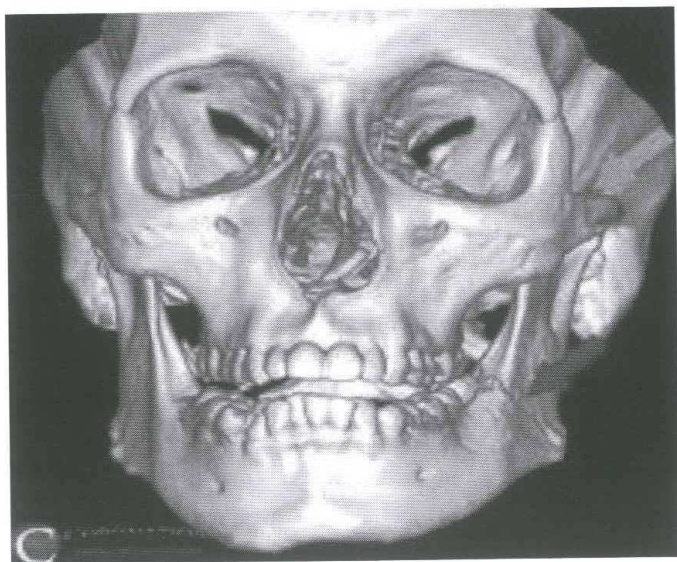
- CT scan images are the imaging modality of choice for facial fractures.
- CT imaging is superior to plain films for delineating multiple fractures, evaluating associated cartilaginous or soft tissue injury, and assessing for the presence of impingement into the optic canal.
- Three-dimensional CT scans are highly recommended for the treatment planning of fractures of moderate or greater complexity.

6. MANDIBULAR FRACTURE

- Although traditionally the mandible is thought to form a complete bony ring, interrupted only by the temporomandibular joints, isolated fractures are relatively common occurring in about 40% of cases.
- The **mandibular condyle** is the commonest location for a mandibular fracture, accounting for approximately 30% of all mandibular fractures:



- Treatment can be conservative or may involve formal reduction (which can be open or closed). Closed reduction may be supported with intermaxillary fixation or splints.
- **Possible complications of mandibular fracture include:**
 - Osteomyelitis
 - Permanent malocclusion
 - Permanent paraesthesia



III. SHOULDER AND BRACHIAL PLEXUS INJURY

ANATOMY

• THE ROTATOR CUFF

- Stability is mostly conferred by robust neuromuscular control of the rotator cuff group of muscles.
- These arise from the scapula and insert on the tuberosities of the humerus, giving them the mechanical capacity to stabilise the ball in the socket against the pull of the powerful muscles that move the arm.
 - *Supraspinatus*: abduction
 - *Subscapularis*: internal rotation
 - *Infraspinatus and Teres minor*: external rotation

• STERNOCLAVICULAR JOINT

- The clavicle acts as a strut, keeping the upper limb away from the chest.
- The sternoclavicular joint relies on powerful ligaments to prevent displacement, which is therefore relatively unusual.

• ACROMIOCLAVICULAR JOINT

- The clavicle has a complex relationship with the scapula.
- This allows scapular rotation in full abduction, while assisting in maintaining the position of the upper limb.
- It achieves this by the strong conoid and trapezoid ligaments which anchor the clavicle to the coracoid process of the scapula.

• SCAPULO-THORACIC JOINT

- In full abduction, the gleno-humeral joint can only achieve around 90° of abduction at which point the scapula rotates on the chest wall to achieve the remainder of the arc.

• BRACHIAL PLEXUS

- The upper limb is supplied from the C4 to T1 nerve roots.
- Occasionally T2 is also involved.
- The myotomes are as follows:
 - **Shoulder Joint**
 - C4-5: Abduction
 - C7: Adduction
 - **Elbow Joint**
 - C5-6: Flexion
 - C7-8: Extension
 - C6: Pronation/Supination
 - C6-7: Flexion
 - **Wrist Joint and Fingers**
 - C6-7: Extension
 - T1: Abduction/Adduction (small muscles of the hand)

CLINICAL ASSESSMENT

- Generally, be reluctant to diagnose a sprain. A minor sprain may be the diagnosis, but often there is something more significant to find. Always examine the rotator cuff. If it is intact and there is no bony tenderness or neurological impairment, most will resolve.
- Examination should take the form of the traditional sequence of: look, feel, move and image:

- o **Look:** for deformity, swelling, congestion etc.
- o **Feel:** for site and nature of tenderness (eg, bony, diffuse, subacromial space etc). Check for sensory loss.
- o **Move:** passively, then look for the active range of movement. Bear in mind the myotomes if there appears to be any motor loss. Next assess the rotator cuff. Active resisted movements are tested as follows;
 - **Supraspinatus** abduction 20-40° in extreme internal rotation with palms outwards
 - **Subscapularis** extending hand away from back
 - **Infraspinatus** and **Teres minor** external rotation

INVESTIGATION STRATEGIES

- **Plain Radiographs**
 - o Always ask for 2 views, ie AP, and a modified axial. The modified axial is preferable to the often-used Y view as it is less likely to be misinterpreted.
- **Ultrasound**
- **Computed Tomography (CT)**
- **Bone scintigraphy**
 - o Bone scintigraphy is a highly sensitive method for demonstrating bone pathology, particularly covert fractures and bone metastases.
- **Magnetic resonance (MRI)**
 - o MRI has the advantage of high contrast resolution, making it particularly useful for the assessment of soft tissue injuries.

1. ACROMIO-CLAVICULAR DISRUPTION

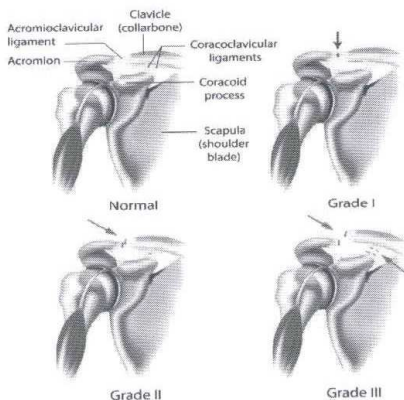
• Mechanism of injury:

- o Dislocations of the acromioclavicular joint are common
- o Usually being caused by a fall onto the point of the shoulder.

• Clinical:

- o There is an apparent upward displacement of the end of the clavicle, though it is the whole of the shoulder girdle on the affected side, which has sagged to leave the clavicle prominent.
- o This is due to *rupture of the coracoclavicular ligaments which have a suspensory function for the upper limb.*

AC Joint Sprain / AC Joint Separation



• Management:

- o The majority recover to a reasonable degree of function in **3 weeks** with a **simple sling** and **analgesia**, and only the **most severe need surgery**.
- o In the ED, do not confuse this injury with a distal clavicular fracture: an xray may be helpful.
- o Stress x-rays are not required in the majority of cases, and may even look normal, due to the upward pull of the trapezius on the scapula.

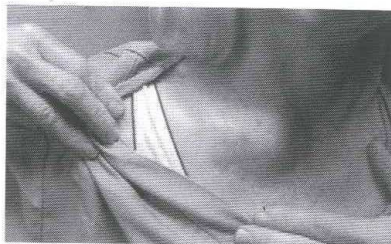
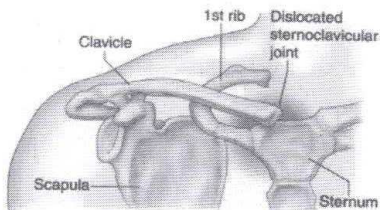
2. STERNO-CLAVICULAR DISRUPTION

• Mechanism of Injury:

- o Direct force to the front of this joint can cause a posterior dislocation, one of the few upper limb injuries that can cause an immediate threat to life.

• Clinical:

- o Severe pain
- o Cough/ Hoarseness
- o Pneumothorax
- o Tracheal compression.
- o There may be venous congestion due to compression of the **internal jugular vein**, along with ipsilateral arm venous congestion.
- o The medial end of the clavicle is usually easily palpated but with a posterior dislocation it has disappeared on the affected side.



• Imaging:

- o The sternoclavicular joint is notoriously difficult to interpret on plain X-rays and in cases of airway compromise clinical assessment alone may be all that is required before treatment is attempted.

• MANAGEMENT

o Analgesia

- o An attempt can be made at **closed reduction**. Traction is applied to the arm and it is sometimes possible to grasp the clavicle through the skin and pull it forwards, hopefully resulting in a pop as reduction occurs.
- o In extremis, the traditional method of bringing the clavicle away from the trachea is to grasp it with a towel clip through the skin and pull forwards.

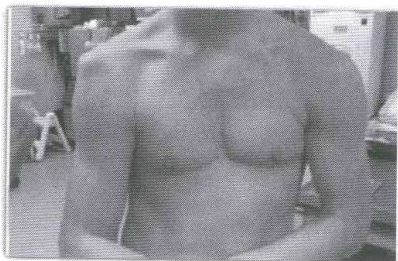
3. ANTERIOR/ INFERIOR SHOULDER DISLOCATION

• Mechanism of injury:

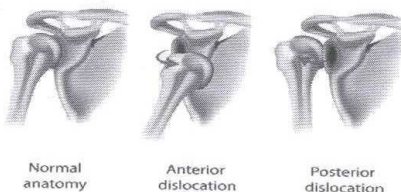
- o Common, often resulting from **forced external rotation of the upper limb**.

• Clinical:

- o From the rear, the shoulder assumes a "**Cows bottom appearance**" clinically as the acromion becomes the most lateral aspect of the shoulder.



Shoulder Dislocation



Normal anatomy

Anterior dislocation

Posterior dislocation

Imaging:

- o The xray is typical.
- o In the axial view a lesion is often seen in dislocation, known as a **Hill-Sachs deformity**.
- o This always occurs in recurrent dislocations, and tells the treating physician that at least one previous dislocation has occurred.

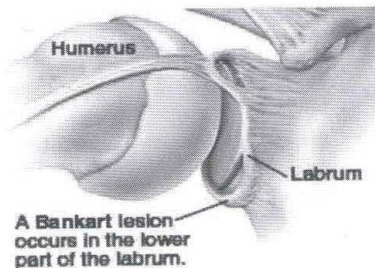


Anterior Glenohumeral Dislocation



*Anterior shoulder dislocation on X-ray with a large **Hill-Sachs lesion***

- o Another lesion tends to occur as the shoulder dislocates because it tears the anterior labrum, especially in younger patients.
- o The tear is usually to lower part of the labrum, and this is called a **Bankart lesion** (Sometimes a tear develops in the upper labrum, often referred to as a superior labral antero-posterior tear (or **SLAP lesion**), though this is often due to sports injuries and not dislocation).



- **MANAGEMENT**

- o The sooner these injuries are diagnosed the easier they are to **reduce**.
- o This can often be achieved under **entonox alone**.
- o There are a variety of reduction methods in popular use, though evidence from clinical trials supporting one method over another is lacking:
 - **The Hippocratic method:** is safe, provided that any counter traction does not apply local force in the axilla.
 - **The Spaso manoeuvre:** the upper limb is held externally rotated by the body, in traction, then gradually flexed through, if necessary, 180 degrees.
 - **The classic Kochers manoeuvre:** risk of intra-articular or spiral humeral shaft fracture.
- o **Arm sling for three weeks.**

4. POSTERIOR SHOULDER DISLOCATION

- **Mechanism of injury:**

- o Relatively rare but are often missed as they can look surprisingly normal.
- o Associated with **epileptic seizures, electrocution** and the **obtunded state** of these patients in the Emergency Department may contribute to the miss.
- o They occur with forced **internal rotation and adduction of the shoulder** and characteristically the patient **loses the ability to externally rotate**.
- o **The light bulb sign** seen on an AP view of the shoulder is characteristic.

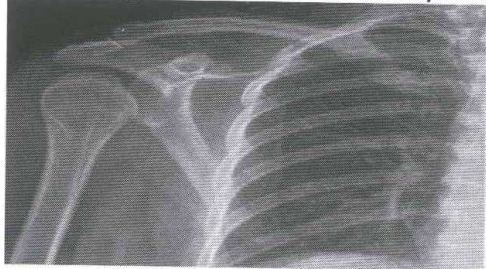
- **Imaging:**

- a. Anteroposterior (AP) view**



- This dislocation may be difficult to appreciate on an AP view because it is not inferiorly displaced and may appear to be in the glenoid fossa. Note that the space between the glenoid fossa and the humeral head does not look normal. The scapular Y view, right, reveals that a posterior dislocation is present. Note that the humeral head lays posterior to the glenoid fossa rather than being centered over it. These injuries are seen best in the **axillary view** (or modified axial), and failure to take this view will inevitably result in missed injuries, and consequent litigation.

- b. Posterior shoulder dislocation seen on a scapular Y view**



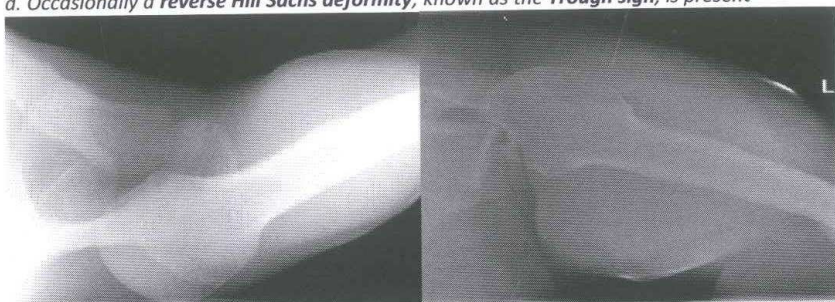
- The anteroposterior view does not definitively show the dislocation. No superior or inferior displacement of the humeral head is seen because the dislocation is directly posterior. The head of the humerus appears to maintain a normal relationship with the glenoid fossa and the acromion process on superficial observation.
 - Definite abnormalities exist on this film, however. The space between the humeral head and the glenoid fossa is abnormal, and the head and neck are seen end on and resemble a **light bulb** because of the extreme internal rotation of the humerus. It becomes obvious on the **Y view**, right, that the humeral head is posteriorly dislocated. It projects posteriorly under the scapular spine rather than in its normal location, centered over the glenoid fossa

- c. Anteroposterior views comparing posterior dislocation, left, and a normal shoulder joint, right.**



- Posterior shoulder dislocation causes internal rotation of the humeral head, which makes the head appear as a **light bulb** rather than its normal club-shaped appearance. Note that the space between the articular surface of the humeral head and the anterior glenoid rim is also widened, and the overlap between the head and the fossa is decreased.

d. Occasionally a **reverse Hill Sachs deformity**, known as the **Trough sign**, is present



- Posterior shoulder dislocations are associated with **seizures** and **electrocution**, and the incidence of bilateral posterior dislocations is higher than bilateral anterior dislocations, which are very rare, though they do occur following seizures.
- For this reason, a low threshold for xray investigation of both shoulders should be maintained in these clinical settings.

5. CLAVICLE FRACTURE

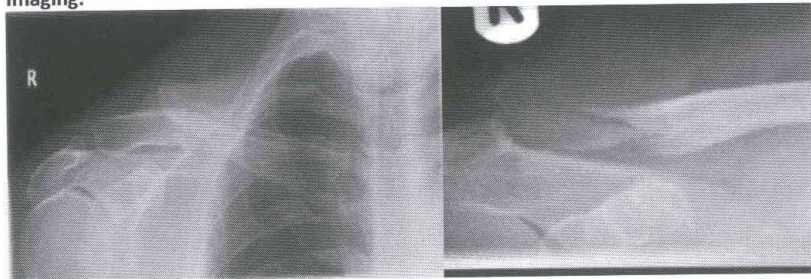
• Mechanism of injury:

- The clavicle is one of the most common bones to be fractured, most often in the middle third.
- Children are particularly prone to the fracture, and newborns may present with a clavicle fracture following a difficult delivery.

• Clinical:

- The sterno-mastoid raises the proximal part of the fracture, and the weight of the upper limb causes the shoulder to drop.

• Imaging:



- In this image that clavicle fracture is obvious. However, the associate fractured ribs and pneumothorax were missed, as the eye is readily drawn to one injury, resulting in the assumption that no other injury is present.
- Pathological fractures are not uncommon in the clavicle. In this image, the moth-eaten nature of the bone is obvious.

• MANAGEMENT

○ Uncomplicated fracture:

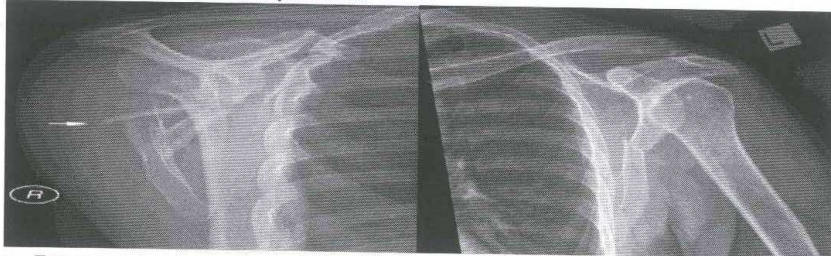
- Sling,
- Analgesia, and allow the bone to heal itself,
- Monitoring progress with X-rays every week or few weeks.

o Indications of surgical repair

- Comminution with separation
- Significant shortening of the clavicle
- Skin penetration
- Associated neurological or vascular injury
- Non-Union after 3-6 months

6. SCAPULAR FRACTURE

- The scapula is sturdy and located in a protected place, so it rarely breaks.
- When it does, it is an indication that the individual was subjected to a considerable amount of force and that a chest injury be present. Direct falls on to the back, especially involving force are the most common cause, such as being thrown from a horse. The injury may not be noticed because it may be accompanied by other, more severe injuries.
- Diagnosis may require a **skyline view**



- Treatment involves **pain control** and **immobilizing** the affected area, and, subsequently **physiotherapy**.

7. WINGING SCAPULA

• OVERVIEW

- o The **scapula (shoulder blade)** is the largest bone of the shoulder complex and has the greatest number of **muscles** attached to it.
- o These muscles both stabilise the arm to the body and move the arm around in space.
- o All these muscles act at the same time sometimes and oppose each other at other times, but work together like a well-trained team to allow the arm to move in space.
- o If any of these muscles are not working in the right way at the right time this leads to a break in the rhythmic motion of the scapula. This is known as a scapula **'dysrhythmia'**. This leads to apparent **'winging' of the scapula**.
- o **Winging of the scapula** is a surprisingly common physical sign, but because it is often asymptomatic it receives little attention.
- o Diagnosis is essentially clinical and should be considered in any patient presenting with **shoulder pain or weakness**, as delay in recognition may cause permanent disability
- o Winging may be caused by injury or dysfunction of the muscles themselves or the nerves that supply the muscles.

• CAUSES

- o **Serratus anterior muscle dysfunction**: traumatic injury to the nerve supplying the serratus anterior muscle, **the long thoracic nerve**; or due to damage to the nerve from pressure lesions or a neuritis (inflammation of the nerve).

- Lesion of the accessory nerve or the dorsal nerve of the scapula, affecting the trapezius or rhomboids, respectively.
- Important etiologies causing nerve palsy include compression injury, trauma, vigorous exercise causing traction, or viral illnesses.
- At times the cause may be idiopathic.
- **Guillain-Barré syndrome** can rarely present with winging of the scapula as the first symptom/sign.
- The test for identifying a long thoracic nerve injury is the '**serratus wall test**': *The patient is asked to face a wall, standing about two feet from the wall and then push against the wall with flat palms at waist level.*

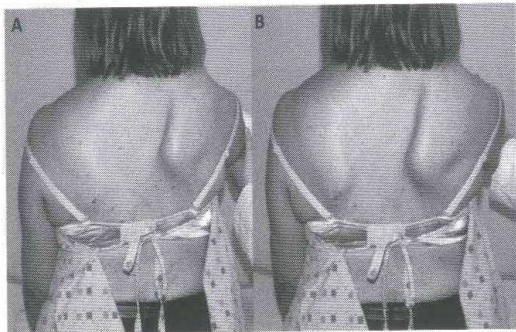


Figure 4.8.13. Winging of scapula

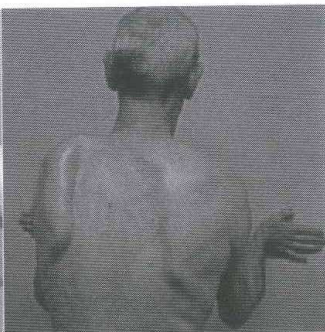


Figure 4.8.14. Wall test for serratus anterior

- A majority of patients respond to conservative treatments involving **physical therapy and range of motion exercises**.
- If conservative treatment fails over the course of 6 months to 1 year, surgical intervention may be considered:
 - **Exploration and decompression of the nerve** can be performed, where it gets trapped or damaged at the scalene muscles in the neck.
 - For more advanced cases, **pectoralis muscle transfer** can be performed.

8. HUMERAL NECK FRACTURE

- The **surgical neck of the humerus** is so called because it is the area of the neck where fractures occur, rather than the anatomical neck.
- A fracture in this area may cause **damage to the axillary nerve**.



- *The surgical neck fracture has resulted in complete separation of the fragments, with a greater tuberosity fracture. The associated axillary nerve injury did not recover despite early surgery.*

9. SLIPPED UPPER HUMERAL EPIPHYSIS

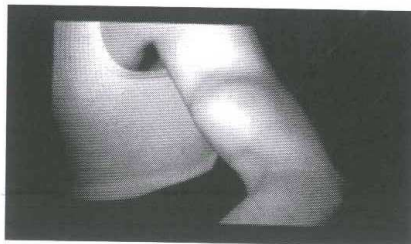
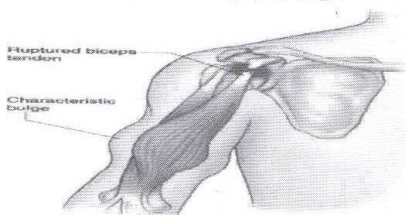
- This is a child/adolescent injury, seen most often between ages of 11 and 15 years.
- The majority are, in fact, **Salter I or II fractures**.
- Occasionally an associated brachial plexus injury occurs.
- They may give the clinical appearance of anterior dislocation, but have a typical xray appearance.



- **Management:**
 - **Less than 5 years of age:** Conservative management.
 - **From 5 to 12 years:** closed reduction may be required for significantly displaced or angulated fractures in children near the end of growth.
 - Operative treatment is rarely indicated. An injury associated with a neurovascular complication is an indication for surgical treatment.

10. BICEPS TENDON RUPTURES

- Rupture of the long head of the biceps tendon leads to bunching of the muscle lower in the arm the so-called **popeye sign**.



- It occurs through a degenerate tendon in the upper part of the bicipital groove.
- **In the majority of cases surgical repair is not indicated.**
- **Distal biceps rupture**
 - Occur in a younger age group and usually have no premonitory symptoms.
 - The rupture typically occurs during a strong contraction and the tendon avulses from its usual point of insertion on the radial tuberosity.
 - The injury is commoner in those who have taken **anabolic steroids**, with muscle strength developing faster than tendon strength.
 - **Repair is important**, and is much easier if it is carried out within 2 weeks or so of injury.
 - If repair is not carried out patients tend to notice poor supination, which is perceived as weak.

11. BRACHIAL PLEXUS INJURIES

- The brachial plexus may be injured in severe distracting injuries to the shoulder, particularly when the shoulder is forced caudally.
- This is typically seen in rugby injuries, falls from horses and falls from motorcycles.
- Success in the repair of proximal brachial plexus injuries is well documented. More distal, the axillary nerve is particularly vulnerable as it winds around the neck of the humerus.

A. AXILLARY NERVE INJURY

- Associated with **humeral neck fracture** and **Dislocated shoulder**.
- It can result in paralysis of the **teres minor muscle** and **deltoid muscle**; so that abduction of the shoulder is impaired and loss of sensation over a small part of the lateral upper arm.

B. ERB'S PALSY

- **Upper Brachial Plexus Injury** (Erb's palsy) commonly occurs where there is excessive increase in the angle between the neck and shoulder, this stretches (or can even tear) the nerve roots, causing damage.
- It can occur as a result of a difficult birth or shoulder trauma.
- **Nerves affected:**
 - Nerves derived from solely **C5-C6 roots**; **Musculocutaneous, Axillary, Suprascapular** and **Nerve to Subclavius. (MASS nerves)**
- **Muscles paralysed:**
 - **Supraspinatus, infraspinatus, subclavius, biceps brachii, brachialis, coracobrachialis, deltoid and teres minor.**
- **Motor functions:**
 - The following movements are lost or greatly weakened (**FALS**):
 - **Flexion** at shoulder.
 - **Abduction** at shoulder,
 - **Lateral rotation** of arm,
 - **Supination** of forearm,
- **Sensory functions:**
 - Loss of sensation down lateral side of arm, which covers the sensory innervation of the axillary and musculocutaneous nerves.
 - The affected limb hangs limply, **medially rotated** by the unopposed action of pectoralis major. The forearm is **pronated** due to the loss of biceps brachii.
 - This is position is known as '**waiter's tip**', and is characteristic of **Erb's palsy**.

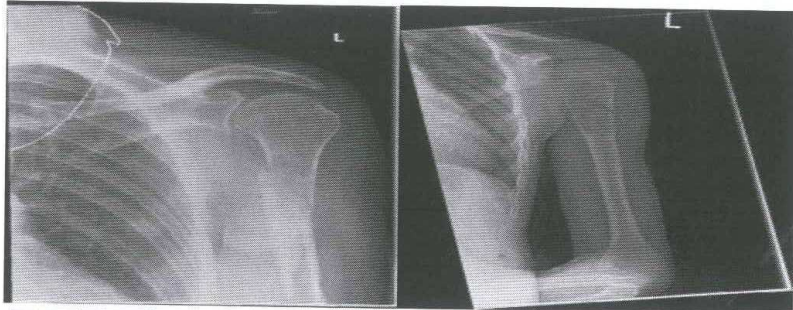
C. KLUMPKE'S PALSY

- **Lower Brachial Plexus Injury** results from excessive abduction of the arm (e.g. person catching a branch as they fall from a tree).
- It has a much lower incidence than Erb's palsy.
- **Nerves affected:**
 - Nerves derived from the **C8-T1 root: Ulna and Median Nerves.**
- **Muscles paralysed:**
 - All the small muscles of the hand.
 - Note that the flexors muscles in the forearm are supplied by the Ulna and Median Nerves, but are innervated by different roots.

- **Sensory functions:**
 - Loss of sensation along medial side of arm.
 - The classic presentation of Klumpke's palsy is the "**claw hand**" deformity.
 - There is associated **Horner syndrome** if there is involvement of the cervical sympathetic chain.
- **Horner syndrome** results from an interruption of the sympathetic nerve supply to the eye and is characterized by **Miosis** (ie, constricted pupil), **Partial Ptosis**, and Loss of Hemifacial Sweating (ie, **Anhidrosis**) and **Enophthalmos**.

12. PATHOLOGICAL FRACTURE

- The commonest causes are:
 - Simple bone cysts,
 - Fragility fractures
 - Metastatic lesions,
 - Fibrous dysplasia,
 - Giant cell tumor of bone
 - Multiple myeloma



- **Bone scintigraphy** may reveal other bone lesions which are not evident from radiographs, (but multiple myeloma is not usually hot).
- Sometimes it is clear that a fracture is pathological, but the cause is not immediately clear.



No cause

Multiple myeloma

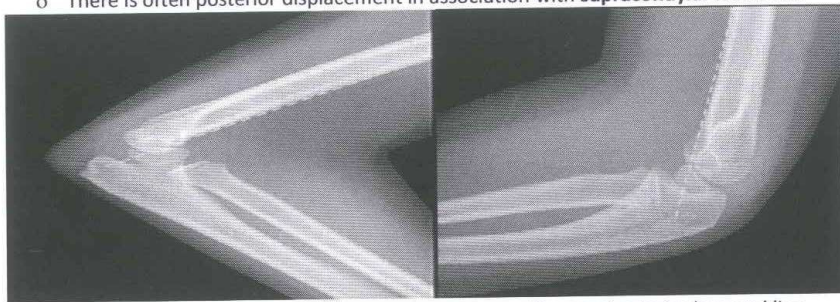
Metastasis

IV. ELBOW INJURIES

IMPORTANT RADIOLOGICAL LINES

1. ANTERIOR HUMERAL LINE

- If a line is drawn along the anterior part of the humerus on the lateral radiograph, then it should intersect the middle third of the capitellum.
- Failure to do this indicates that the capitellum has been displaced.
- There is often posterior displacement in association with **supracondylar fractures**.



Normal anterior humeral line

Abnormal anterior humeral line

2. THE RADIO-CAPITELLAR LINE

- A line drawn through the middle of the radius should always bisect the capitellum since the radial head articulates with the capitellum.
- This should occur in every direction, no matter which x-ray view is taken.
- If this is not the case **suspect dislocation of the radial head** and remember that this can sometimes be associated with ulna fractures (**Monteggia fracture-dislocation**).

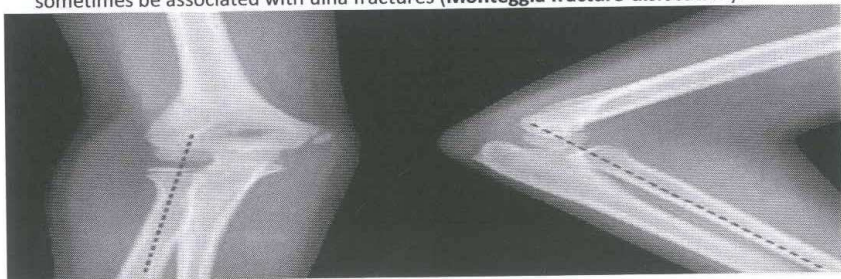


Figure 7: Normal radio-capitellar line



Normal AH & RC lines

Abnormal Radio-capitellar line

3. FAT PADS

- On a normal AP x-ray of the elbow an anterior fat pad is visible due to fat in the joint capsule. *It is **never normal** to see a posterior fat pad because this is hidden in the intercondylar fossa.*
- The diagram below shows a **displaced anterior fat pad in association with a posterior fat pad**.
 - This is known as the **Sail sign** because of its resemblance to the sails of a boat.
 - In the setting of acute trauma, it represents **blood in the joint**.
 - In the non-trauma setting effusion may be due to an inflammatory cause.
 - Note that if the fracture is extra-articular, then there may not be a joint effusion and therefore the fat pad sign will be absent.
 - Examine the radial head closely in these injuries as there is often a subtle fracture.
 - Presence of a **posterior fat pad** has been associated with a **75% rate of occult fracture**.

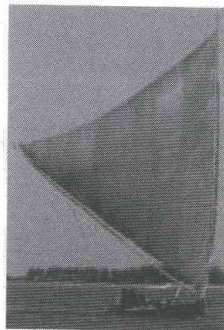


Figure 4.8.15. Positive Sail Sign and Posterior Fat Pad

4. OSSIFICATION CENTRES

- There are six ossification centres in the elbow of a developing child and they occur in a fixed order at sequential times **up to the age of 13 years**, although these times are variable.
- It is the presence of these centres that make paediatric elbow x-rays notoriously difficult to interpret. Knowledge of these ossification centres and the age at which they appear will assist the observer in identifying whether a fracture is present or not.
- A well-known helpful mnemonic for this is **CRITOL** or **CRITOE**:

Capitellum	1 year
Radial head	3 years
Internal (medial) epicondyle	5 years
Trochlear	7 years
Olecranon	9 years
Lateral (External) epicondyle	11 years

- Note that these ages vary but a broad guide of **1, 3, 5, 7, 9 and 11 years** is easy to remember.

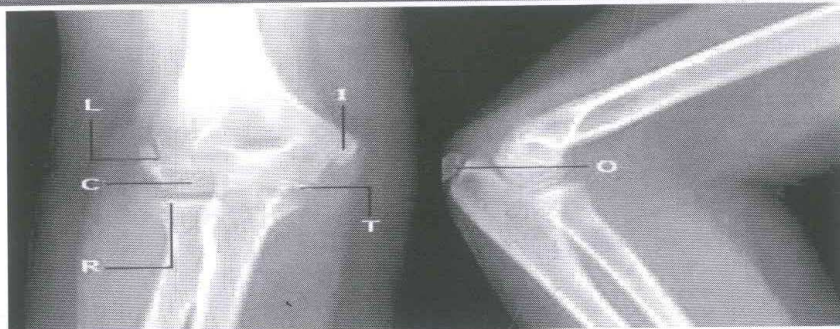


Figure 4.8.16. Ossification centres

5. CONTOURS

- Finally, it is important to analyse all 3 bones of the elbow joint and follow the contours looking for irregularities and steps that could indicate subtle disruptions and fractures in the cortex. Common subtle injuries that may be difficult to identify include **undisplaced radial head fractures in adults** and **undisplaced supracondylar fractures in children**.

MANAGEMENT OF ELBOW INJURIES IN ED

1. ELBOW DISLOCATION

- **Mechanism of injury:**
 - o Seen in both children and adults.
 - o Caused by a **FOOSH** (fall onto the outstretched hand).
 - o It is more common in children than dislocation of the shoulder.
- **Clinical:**
 - o Examination may reveal obvious deformity of the elbow.
 - o The triangular relationship of the epicondyles and olecranon will be disrupted.
 - o It is important to **check the distal neurovascular status** of the limb due to possible damage to the **brachial artery or median and ulnar nerves**.
 - o The dislocation is most commonly in a posterior or posterolateral direction and will be confirmed on x-ray, along with the presence of any associated fractures.
 - o **Associated epicondylar fractures and fractures of the lateral condyle** are known to occur in children.



Figure 4.8.17. Posterior elbow dislocation

• MANAGEMENT

- o **Analgesia** should be provided prior to attempts to reduce the dislocation.
- o **Procedural sedation** (with full monitoring) is likely to be required.

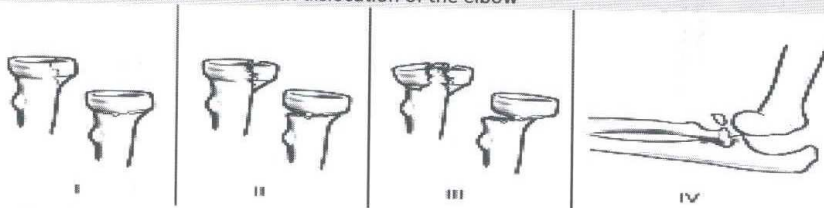
- o In some cases, **reduction under general anaesthetic** may be necessary.
- o Reduction should be immediately followed by a **further assessment of limb neurovascular status**. Successful reduction is then confirmed by **repeat x-ray**. This will also enable assessment of the new position of any associated fractures.
- o The reduced elbow can be immobilised in a **backslab in 90 of flexion**.
- o Admit for observation where there are concerns over neurovascular impairment or significant elbow swelling.
- o **Outpatient orthopaedic** review should subsequently be arranged.
- o **Myositis ossificans** may later develop as a result of large elbow haemarthroses

2. RADIAL HEAD FRACTURES

- **Mechanism of injury:**
 - o These injuries usually follow a fall onto an outstretched wrist or direct trauma.
 - o Usually occur in adults and account for 30% of all adult elbow fractures.
- **Clinical**
 - o Local bruising and swelling.
 - o In some cases, pain may only be evident with palpation of the radial head during passive forearm pronation.
 - o Elbow extension is usually restricted.
 - o Assessment of the wrist should be performed due to the possibility of an **Essex-Lopresti fracture-dislocation**, consisting of a comminuted radial head fracture with subluxation of the distal end of the ulna.

MASON JOHNSTON CLASSIFICATION OF RADIAL HEAD FRACTURE

- o I - Nondisplaced
- o II - Minimally displaced with depression, angulation and impaction
- o III - Comminuted and displaced
- o IV - Radial head fracture with dislocation of the elbow



- **Imaging:**
 - o Radiography may reveal the fracture or merely the presence of a joint effusion.

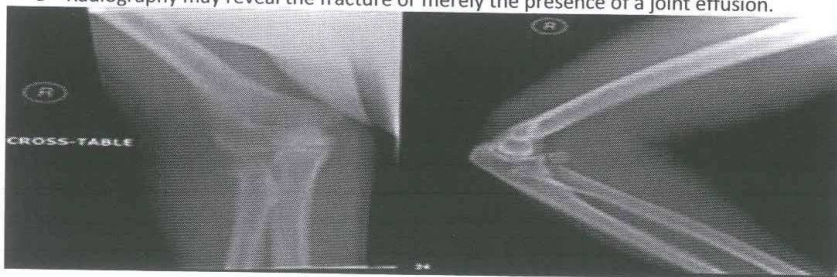


Figure 4.8.18. Radial head fracture

- **Management:**
 - **Undisplaced fractures:**
 - Collar and cuff sling and orthopaedic outpatient follow-up.
 - Early mobilization to prevent loss of elbow extension.
 - **Comminuted or displaced fractures:**
 - Manipulation under anaesthetic or internal fixation.
 - Occasionally the radial head may need to be excised and replaced.
 - **Radial neck fractures:**
 - Seen more commonly in children and are managed similar to radial head fractures.
 - Greater than 20° of angulation in the adult requires reduction.

3. OLECRANON FRACTURES

- **Mechanism of injury:**
 - Fall onto the point of the elbow or onto a semi-flexed outstretched forearm.
- **Clinical:**
 - Swelling and tenderness over the posterior aspect of the elbow.
- **Imaging:**
 - Radiography will confirm the diagnosis and also reveal any displacement due to the pull of the triceps tendon.
 - Identification of paediatric olecranon fractures may be complicated by the appearance of the olecranon ossification centre, which may be bifid.



Figure 4.8.19. Olecranon Fracture

- **MANAGEMENT**
 - **Undisplaced fractures:** backslab in 90° of elbow flexion and orthopaedic clinic follow-up.
 - **Displaced fractures (> 2mm)** and **those with comminution:** are more likely to require operative fixation and therefore warrant orthopaedic referral.

4. MONTEGGIA FRACTURE-DISLOCATION

- **Mechanism of injury:**
 - This injury comprises **dislocation of the radial head with an ulna fracture (GRUM)**
 - It may result from a direct blow to the ulna or forced pronation.
- **Imaging:**
 - The radiographic appearance of a dislocated radial head (suspect if a line bisecting the radius longitudinally does not pass through the centre of the capitellum) should prompt further imaging of the forearm to exclude an ulna fracture.

- **Management:**

- o These injuries should be referred for reduction and internal fixation.



Figure 4.8.20. Monteggia fracture

5. GALEAZZI FRACTURE-DISLOCATION

- **Mechanism of injury:**

- o This injury comprises **dislocation of the distal radioulnar joint with a Radial fracture (GRUM)**.
- o Fall on an outstretched hand (FOOSH) with a flexed elbow
- o Galeazzi fractures are classified according to the position of the distal radius:
 - **Type I:** dorsal displacement
 - **Type II:** volar displacement

- **Imaging:**



Figure 4.8.21. Galeazzi fracture

- o Plain films are usually sufficient for diagnosis and management planning, however good quality orthogonal views are needed to correctly identify and characterise displacement.

- **Management:**

- o These injuries should be referred for reduction and internal fixation.

6. PAEDIATRIC INJURIES

A. SUPRACONDYLAR FRACTURES

- **Mechanism of injury:**

- o 60% of all paediatric elbow fractures. Follow a fall onto an outstretched hand.
- o The peak incidence occurs between the ages of **5 and 8 years**.
- o Children are more prone to supracondylar fracture than adults due to the relatively thin trabeculae of the coronoid and olecranon fossae in this population.
- o 90% of these fractures are caused by hyperextension injury due to ligament laxity.
- o The force is transmitted up through the ulna and into the distal humerus.

- **Imaging:**

- Presence of **fat pads** and **loss of normal anterior humeral alignment**.
- Undisplaced fractures may only be identified by subtle disruption in the posterior cortex whereas displaced fractures will normally be obvious.

GARTLAND CLASSIFICATION OF SUPRACONDYLAR FRACTURES

Type 1	Undisplaced Fracture
Type 2	Anterior displacement with an intact Posterior Cortex.
Type 3	Complete cortical disruption with anterior and posterior displacements.

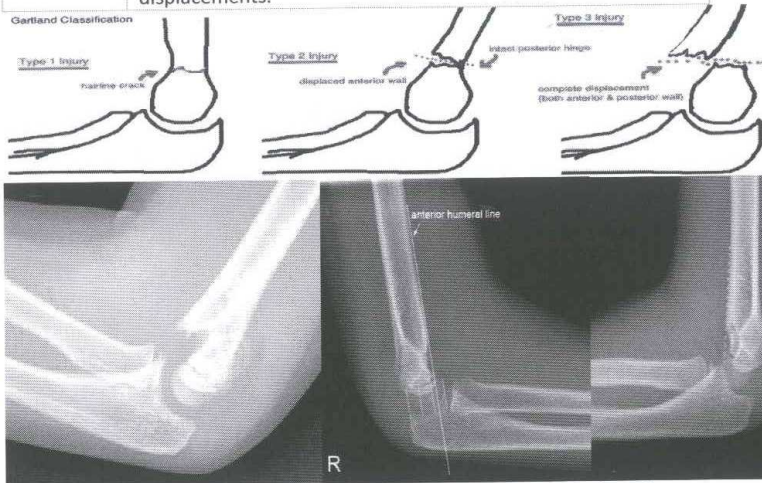


Figure 4.8.22. Supracondylar fractures with posterior fat pad

- **Management:**

- Analgesia and a search for associated neurovascular complications.

- **Undisplaced fractures:**

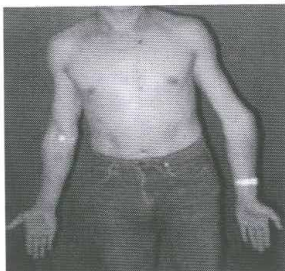
- Collar and cuff and can be followed up in fracture clinic.
- If there is significant pain, a back slab may be a better option.

- **Displaced fractures:**

- Should all be referred for manipulation,
- Urgently if circulation is compromised.

- **Complications include:**

- *Cubitus varus (Gun stock deformity)*
- *Malunion and stiffness*
- *Myositis ossificans*
- *Nerve injury (most commonly median nerve)*
- *Brachial artery injury (due to stretch and posterior displacement)*
- *Volkman's ischaemic contracture (due to compartment swelling)*



Gun stock deformity



B. LATERAL EPICONDYLE AVULSION INJURY

- **Mechanism of injury:**

- This is the second most common elbow fracture seen in children, usually between the ages of **4 and 10**.
- It results from a varus force applied through the extended elbow, normally due to a fall on an outstretched hand.
- It is commonly displaced **by the action of the forearm extensors**.

- **Imaging:**

- Appearance on x-ray may be subtle so an awareness of the possibility of this injury is necessary when interpreting films.

- **Management:**

- **Undisplaced fractures:** Backslab with orthopaedic follow up.
- **Displaced fractures:** often need reduction



The AP view demonstrates displacement of the lateral epicondyle in keeping with an avulsion fracture (arrow).

On the lateral view, multiple fracture fragments are seen within the joint space (arrow). The AP view shows avulsion of the lateral epicondyle

C. MEDIAL EPICONDYLE AVULSION INJURY

- These injuries tend to occur in adolescents due to valgus stress during a fall on an outstretched hand. There may be associated **ulna nerve** damage and sometimes dislocation. Undisplaced avulsions can be managed conservatively while displaced fragments should be referred for reduction.

D. RADIAL NECK FRACTURE (CHILD)

- These injuries are more common in children due to weak metaphyseal bone and, as with radial head fractures, may be difficult to spot on an x-ray.
- Treatment is similar to that of radial head fractures and orthopaedic referral is recommended if there is greater than 30 degrees of angulation.

E. PULLED ELBOW

- This is also sometimes known as "**Nursemaids Elbow**".
- There is often a history of traction on the arm of a child between **1 and 5 years**, although this is not always the case. The parent may not be willing to volunteer the history or may not have been present when it occurred. The child will not be using the arm.
- It results from **subluxation of the radial head** from its normal position encircled by the annular ligament. The x-ray is normal and therefore not necessary if clinical suspicion is high prior to attempted manipulation.
- Traditional reduction is achieved by **flexing the elbow to 90 degrees and then fully supinating or pronating the forearm**, there may often be an associated click and the child will begin using the arm a short time later.

V. WRIST INJURIES

1. UNDISPLACED FRACTURE OF DISTAL RADIUS

A. GREEN STICK FRACTURE

- These are partial fractures, since only one part of the bone is broken and the other side is bent. The name is derived from an analogy of breaking a young, fresh tree branch.
- Most often the greenstick fracture must be bent back into the proper position.
- Greenstick fractures can take a long time to heal because they tend to occur in the middle, more slowly growing parts of bone.



B. TORUS/BUCKLE FRACTURE

- Torus fractures, or **buckle fractures**, are extremely common injuries in children.
- Because children have softer bones, one side of the bone may buckle.
- The word torus is derived from the Latin word 'Tori' meaning swelling or protuberance. These injuries tend to heal much more quickly than the similar greenstick fractures.

- **Management**

- Backslab and sling and Refer to the Fracture Clinic

2. FRACTURES OF THE ULNAR STYLOID

- No active treatment required; Backslab for comfort, sling and Fracture Clinic

3. SCAPHOID INJURIES

- **Mechanism of injury:**

- Most common carpal fracture (70%), *followed by triquetral and trapezium*
 - The geometry of the scaphoid as it relates to its retrograde blood supply renders it particularly prone to **avascular necrosis** and other fracture complications.
 - FOOSH with **hyper-extension of the wrist**

- **Imaging:**

- Initial x-ray (full scaphoid series): Specificity is 100% but Sensitivity is 80%
 - Pre-Test Probability of scaphoid fracture in patient with scaphoid wrist pain and non-diagnostic x-rays is about **25%** (17-38% across 4 studies) so:
 - 1 out of 4 people with a negative initial x-ray have a fracture
 - Or 3 out of 4 people going home in splint don't have a fracture.

- **Clinical sign:**

- Thumb compression pain
 - Scaphoid tubercle
 - Snuff box tenderness
 - Ulnar deviation pain
 - **Clamp sign:** ask patient "exactly where does it hurt?". The patient will form a clamp with the opposite thumb and index finger on both sides of the thumb.

- o **Pain with resisted supination:** Hold the injured hand with forearm in neutral position. Patient attempts supination = pain when examiner resists

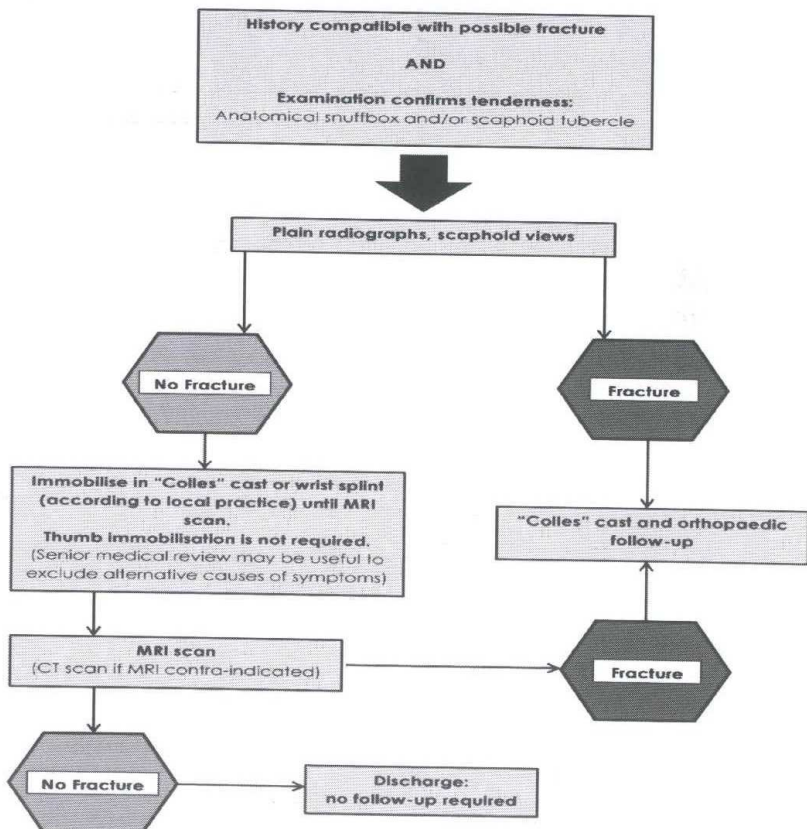


Figure 4.8.23. Scaphoid fracture



Figure 4.8.24. Scapholunate ligament rupture (Terry Thomas)

- **Management:**



- o Radiologically confirmed fractures should be treated by **Colles cast and referral to the Fracture Clinic.**
- o **Refer for ORIF if:**
 - More than 1mm displacement of fragments,
 - Angulation of 15%,
 - Fracture comminution
- o Check x-rays for signs of **ruptured scapholunate ligament (Terry Thomas sign)**
- o If seen, confirm again no evidence of carpal dislocation and treat as a scaphoid fracture.

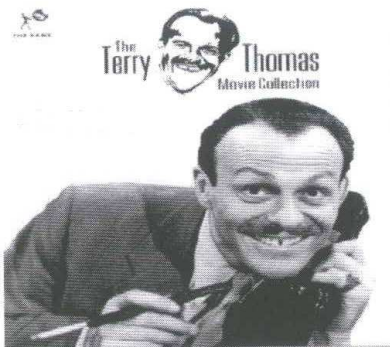
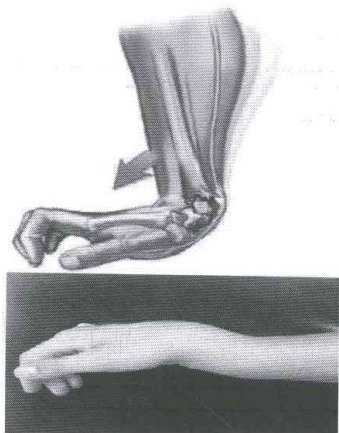


Figure 4.8.24. Terry Thomas sign: rupture of scapholunate ligament

4. COLLES' FRACTURE

- **Mechanism of injury:**
 - o Most Colles fractures are secondary to a fall on an outstretched hand (FOOSH) with a pronated forearm in dorsiflexion (the position one adopts when trying to break a forward fall).
- **Clinical:**
 - o They consist of a fracture of the **distal radial** metaphyseal region with **dorsal angulation and impaction**, but without involvement of the articular surface.
 - o Colles fractures are the most common type of distal radial fracture and are seen in all adult age groups and demographics.
 - o They are particularly common in patients with osteoporosis and as such, they are most frequently seen in **elderly women**.
 - o The relationship between Colles fractures and osteoporosis is strong enough that when an older male patient presents with a Colles fracture, he should be investigated for osteoporosis because his risk of a hip fracture is also elevated.
 - o Manipulate as instructed under **Bier's block or GA** by disimpaction, flexion, pronation and ulnar deviation.
- **Imaging:**
 - o AP and lateral wrist x-rays usually suffice.
 - o The fracture appears extra-articular, and usually proximal to the radioulnar joint.
 - o Dorsal angulation of the distal fracture fragment is present to a variable degree (as opposed to **volar angulation of a Smith fracture**).
 - o There is also usually impaction with resultant shortening of the radius.
 - o An associated ulnar styloid fracture is present in up to 50% of cases.



• Complications of Colle's fracture:

- Malunion resulting in **Dinner fork deformity**
- **Median Nerve Palsy**
- Post traumatic **carpal tunnel syndrome**
- **Reflex Sympathetic Dystrophy**
- **Secondary Osteoarthritis**, more frequently seen in patients with intra-articular involvement
- **EPL tendon tear**

• Management:

○ If stable:

- Apply POP backslab and sling.
- Refer to the Fracture Clinic

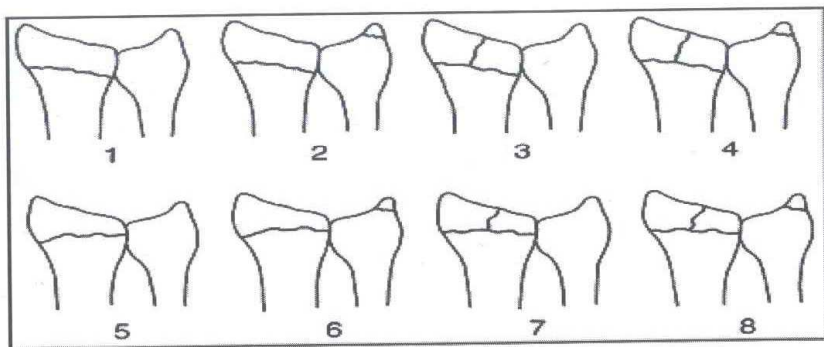
○ Refer to ortho if:

- More than 10° dorsal angulation (tilt)
- Radial shortening more than 3 mm
- Radial shift more than 2 mm
- Dorsal displacement more than 2 mm
- These "rules" may not apply in some (elderly e.g.) patients

FRYKMAN CLASSIFICATION OF DISTAL RADIAL FRACTURES

- It is based on the AP appearance and encompasses the eponymous entities of Colles fracture, Smith fracture, Barton fracture, chauffeur fracture etc.
- It assesses the pattern of fractures, involvement of the radio-ulnar joint and presence of distal ulnar fracture.
- Although it appears complicated, it is actually only a 4-type classification (odd numbered types) with each type having a subtype which includes ulnar styloid fracture (these are the even numbered types).
 - **Type I:** transverse metaphyseal fracture
 - This includes both a Colles and Smith fracture as angulation is not a feature
 - **Type II:** type I + ulnar styloid fracture

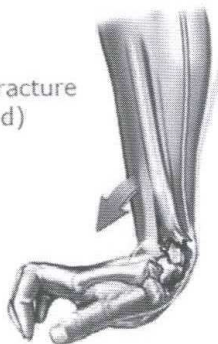
- **Type III:** fracture involves the radiocarpal joint
- This includes both a Barton and reverse Barton fractures
- **Type IV:** type III + ulnar styloid fracture
- **Type V:** transverse fracture involves distal radioulnar joint
- **Type VI:** type V + ulnar styloid fracture
- **Type VII:** comminuted fracture with involvement of both the radiocarpal and radioulnar joints
- **Type VIII:** type VII + ulnar styloid fracture



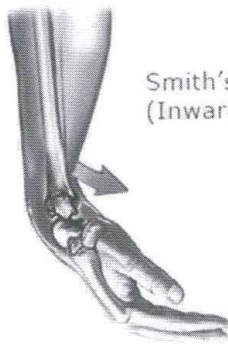
5. SMITH'S FRACTURE

- **Mechanism of injury:**
- Also known as a **Goyrand fracture** in the French literature
 - Fractures of the distal radius with associated palmar (Volar) angulation of the distal fracture fragment.
 - Smith fractures usually occur in one of two ways:
 - A fall onto a flexed wrist
 - Direct blow to the back of the wrist

Colle's Fracture
(Outward)



Smith's Fracture
(Inward)



- Classically, these fractures are extra-articular transverse fractures and can be thought of as a **reverse Colles fracture**.
- The term is sometimes used to describe intra-articular fractures with volar displacement (a **reverse Barton fracture**) or juxta-articular fractures.

Epidemiology

- Smith fractures account for less than 3% of all fractures of the radius and ulna and have a bimodal distribution: **young males (most common)** and **elderly females**.

Imaging:

- In most instances, plain films suffice for diagnosis and characterisation.
- The fracture line is usually evident, although in undisplaced or mildly impacted fractures it can be difficult to see and subtle cortical breaches / buckling should be sought.
- In intra-articular fractures (type II) the degree of articular step-off and gap should be assessed, and this may require CT.

THOMAS CLASSIFICATION OF SMITH FRACTURES

Type I

- Extra-articular transverse fracture through the distal radius
- Most common: ~85%

Type II

- Intra-articular oblique fracture
- Equivalent to a reverse barton fracture
- ~13%

Type III

- Juxta-articular oblique fracture
- Uncommon: <2%

Thomas Classification of Smith Fractures

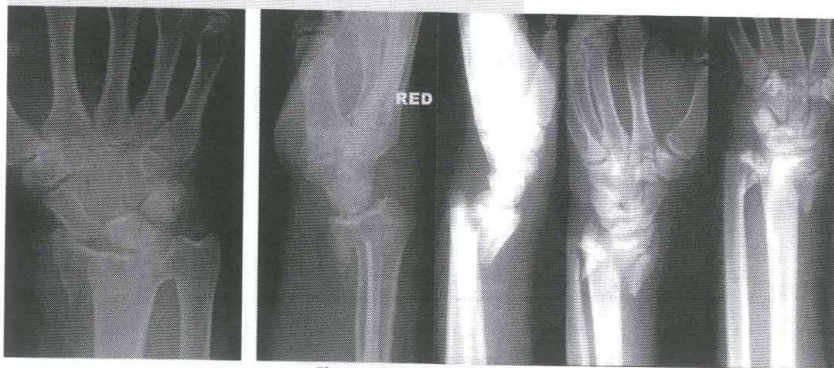
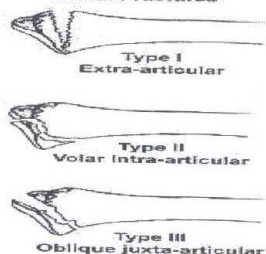


Figure 4.8.25. Smith fractures

Management

- Usually internally fixed and so should be referred to on-call Orthopaedic Team.
- If not manipulate under LA or GA by disimpaction, supination, extension and ulnar deviation and apply ventral POP slab.
- Provide sling and refer to the Fracture Clinic.

6. BARTON'S FRACTURE

- Barton fractures** are displaced intra-articular fracture of the distal radius.

- Volar-type Barton's** is a fracture-dislocation of the volar rim of the radius. This type is the most common.
- Dorsal-type Barton's** is a fracture-dislocation of the dorsal rim of the radius.

- Dislocation of the radiocarpal joint is the hallmark of Barton's fractures.
- These are shear type fractures of the distal radial surface of the radius with translation of the distal radial fragment and the carpus.
- These fractures have a great tendency for redislocation and malunion. They usually require operative treatment.
- **Management:**
 - Often requires MUA under Bier's block.
 - Beware neurovascular compromise - **always check median nerve function** and advise immediate return if symptoms.
 - If reduction not ideal - refer to on-call Orthopaedic Team.

7. CHAUFFEUR'S FRACTURE

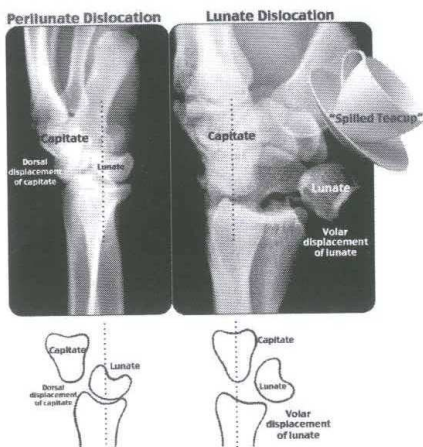
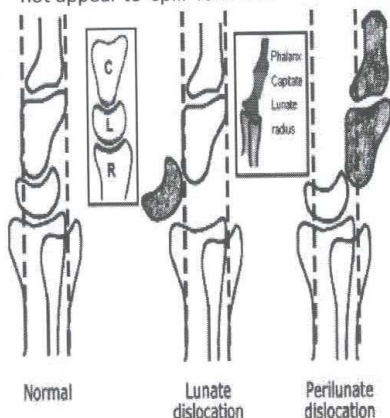
- An isolated fracture of the radial styloid process is also called a **Hutchinson's or chauffeur's fracture**.
- Displacement of the fragment is uncommon.
- There can be associated injury to the scapholunate ligament.
- In most cases a fracture of the radial styloid process is part of a comminutive intraarticular fracture.



8. LUNATE AND PERILUNATE DISLOCATION

8.1. SPILLED TEACUP SIGN

- The **spilled teacup sign** describes abnormal volar displacement and tilt of a dislocated lunate on lateral radiographs of the wrist.
- The convexity of the lunate is no longer in articulation with the distal radius while the concavity is no longer in articulation with the capitate.
- It is an important sign to help differentiate lunate dislocation from perilunate dislocation.
- In the latter, the lunate remains in articulation with the distal radius and therefore does not appear to 'spill' forward.





Spilled Tea Cup Sign of Lunate dislocation

Perilunate dislocation

8.2. PIECE OF PIE SIGN

- The **piece of pie sign** refers to an abnormal triangular appearance of the lunate on an PA image of the wrist indicating lunate dislocation or perilunate dislocation.
- A lateral image will help differentiate whether there is lunate or perilunate dislocation with lunate dislocation demonstrating a spilled teacup sign.

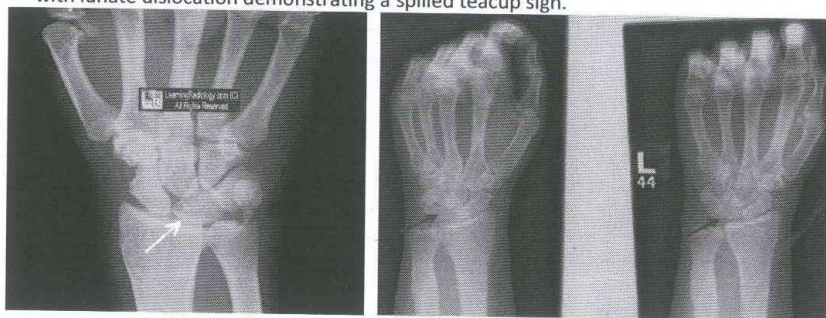


Figure 4.8.26. Piece of Pie Sign: Lunate Dislocation

9. FLAKE TRIQUETRAL FRACTURE

- Immobilise in backslab, sling and refer to the Fracture Clinic.
- Commonest is **flake triquetral fracture** seen on dorsum carpi lateral view.
- Triquetral complication: **Deep branch ulnar nerve**: beware early ulnar motor signs.



Figure 4.8.27. Triquetral fracture seen on dorsum carpi lateral view.

VI. HAND & FINGER INJURIES

1. BENNETT AND ROLANDO FRACTURE-DISLOCATION

- A **Bennett fracture-dislocation** of the thumb results from forced abduction of thumb.
- **Radiographic features**
 - Two-piece fracture dislocation of the base of the thumb
 - Intra-articular
 - Dorsolateral dislocation
 - Small fragment of 1st metacarpal continues to articulate with trapezium
 - Lateral retraction of first metacarpal shaft by abductor pollicis longus
- A **Rolando fracture** is a three part or comminuted intra-articular fracture-dislocation of the base of thumb (proximal first metacarpal). It can be thought of as a **comminuted Bennett fracture**.

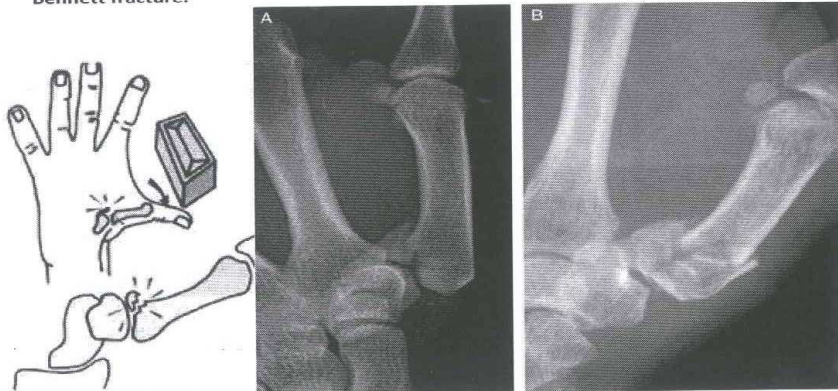
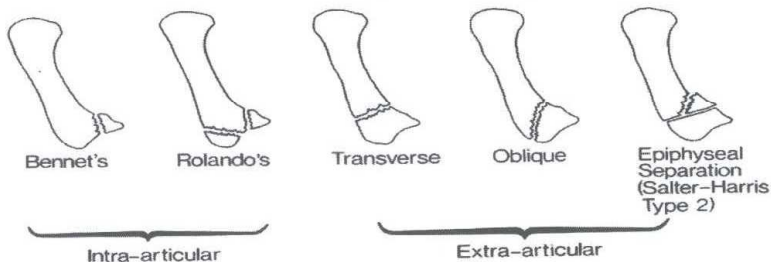


Figure 4.8.28. Bennett Fracture and Rolando fracture



- **Management:**
 - **Treat Bennett's fracture-dislocation with:**
 - Bennett's POP and refer to the on-call orthopaedics.
 - Good initial reduction is important.
 - Thereafter, there is no significant evidence between surgical and conservative management approach.
 - **Fracture base thumb MC not involving joint:**
 - Thumb spica or Bennett's POP and fracture clinic.
 - Sling

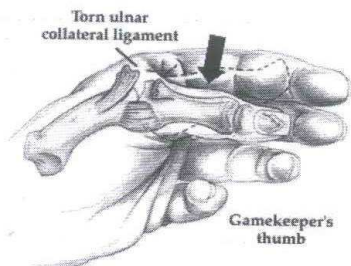
2. GAMEKEEPER'S THUMB

- **Gamekeeper thumb** is essentially synonymous with **Skier thumb**, although the latter has a more acute injury connotation.
- It is an avulsion or rupture of the **ulnar collateral ligament (UCL)** of the thumb.

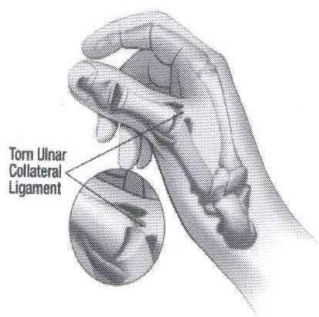
Plain radiograph

- This is almost always the first examination and is often able to give the diagnosis.
- There is a small **avulsion fracture of the ulnar corner of the base of the proximal phalanx**.
- If the tear is in the midsubstance, with no associated fracture then the ulnar side of the joint may appear widened.
- If the diagnosis is suspected stress views were once upon a time recommended, however concern now exists that performing these views can displace the torn undisplaced end of the ligament dorsal to adductor pollicis muscle thereby creating a **Stener lesion**.

Stener lesion: interposition of the adductor pollicis muscle and adductor aponeurosis between torn end of the ulnar collateral ligament and the base of the proximal phalanx



Game Keeper's Thumb



Ultrasound

- Ultrasound is helpful in identifying not only the tear but also whether or not a **Stener lesion is present**. Clearly this requires a knowledge of local anatomy and use of a high frequency probe.

MRI

- MRI is increasingly used to assess x-ray occult injuries to the ulnar collateral or to attempt to identify a **Stener lesion**.

Treatment and prognosis

- Refer to ortho on duty
- Treatment depends on classification, but essentially boils down to whether there is displacement or instability: if there is, surgical fixation is required.
- The presence of a **Stener lesion** an indication for surgery.

3. BOXER FRACTURES

- **Mechanism**

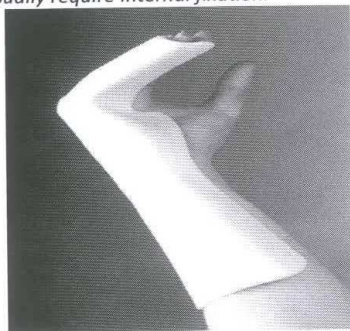
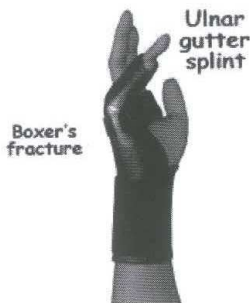
- Boxer fractures are an impaction injury (axial loading of the 5th metacarpal) almost always result as a consequence of a direct blow with clenched fist against a solid surface. They are minimally comminuted, transverse fractures of the 5th metacarpal and are the most common type of metacarpal fracture. They should not be confused with a **boxer knuckle** which represents tendinous and ligamentous disruption of the metacarpal phalangeal joint.

- **Plain radiographs**

- In most cases the fracture is in a transverse plane and minimally impacted.
- It is usually angulated in a volar direction.
- Spiral fractures or angulation in other directions are also sometimes encountered.

- **Treatment and prognosis**

- A short arm **gutter-splint** is applied, with flexion of the metacarpophalangeal joint, typically for 2-3 weeks followed by buddy-strapping.
- Prolonged immobilisation can lead to stiffness.
- **Check rotational deformity**
- Fracture Clinic Follow up
- Severely displaced fractures may benefit from K wire.
- *Fractures of the fourth metacarpal neck can be treated in a similar fashion, whereas the second and third metacarpals usually require internal fixation.*



4. PROXIMAL METACARPAL INJURIES

- Most are stable injuries if sustained with a clenched fist (no rotation at the time of injury) but:
 - **Beware of a proximal (carpometacarpal) dislocation.**
 - Tender over the proximal metacarpal (and carpo-metacarpal area)
 - Deformity may appear subtle. Usually no rotation
 - As with most other limb view **scrutinise the lateral view** for deformity or unusual appearance



Figure 4.8.29. Proximal MCP fracture



Figure 4.8.30. Little MCP fracture

5. DISLOCATION OF PIP AND DIP JOINTS

- Confirm with X-ray (exclude fracture).
- Reduce under LA.
- Assess stability (AP side-to-side).
- **If stable**, apply a **Bedford splint**, **sling** and refer to the fracture clinic.
- **If unstable**, refer to the on-call orthopaedic Team. Always do a post reduction x-ray

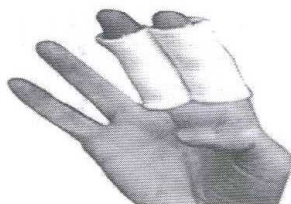
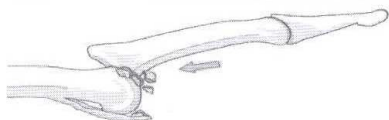
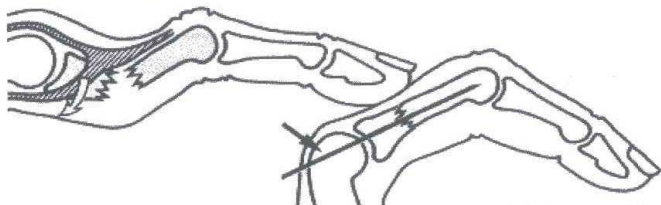


Figure 4.8.31. Bedford splint

6. VOLAR PLATE INJURIES

- Hyperextension injury
- Ligament may avulse chip off base mid phalanx by the volar plate.
- Undisplaced small chips heal without problems
- Larger (> 25% articulation surface) or comminuted fragments best internally fixed
- Check for and document any co-existing collateral ligament damage
- **Neighbour strap** and encourage early joint mobilisation
- Refer to fracture clinic



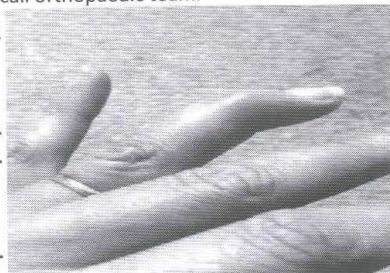
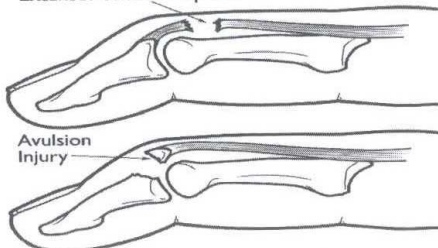
7. PHALANGEAL FRACTURES

- **Transverse fractures** of the proximal phalanges are unstable (often need fixing)
- **Spiral fractures** are also unstable and particularly prone to rotation.
- **ANY rotation deformity** must be corrected and splinted in a position of anatomical function before discharge from the department.
- **All spiral** fractures are followed up in the next fracture clinic.
- Refer to on-call ortho if reduction not achieved
- **Displaced finger** fractures involving the joint should be referred to the orthopaedic team on call.
- **Open fractures** other than those of the tuft require immediate referral.
- Those of the tuft require wound toilet and anti-staphylococcal antibiotics.

8. MALLET FINGER

- X-ray to look for avulsion fracture (better chance of healing).
- With a large fragment check that the DIP joint is not subluxed.
- Check that PIPJ is not going into hyperextension (occurs in small % of those with mallet) - will need to be treated if present.
- **General management includes:**
 - Apply a **mallet (stack) splint** full-time for 6 weeks, then at night for 2 weeks.
 - Ortho/GP/Plastic follow up (depending on your local guidelines).
- Follow 2-week rule - if extension lag at any time then return to splint full time for another fortnight.
- If splint is removed the finger must be kept straight even when washing.
- It is important that the mallet splint allows for full flexion at the PIP joint and patients are encouraged to mobilize at the PIPJ level.
- If the joint is subluxed please refer to the on-call orthopaedic team.

Extensor Tendon Rupture



Top: A rupture of the extensor tendon.

Bottom: A fragment of the distal phalanx has pulled away with the tendon.

9. BOUTONNIÈRE FINGER

- Finger extensor tendon normally has **two lateral slips** (inserting into distal phalanx) and a **middle slip** inserting into the base of the intermediate phalanx.
- If this middle slip ruptures the patient may have point tenderness as the site of the rupture and a "**button hole**" or **Boutonniere deformity** ensues.
- Patients will be unable to extend the PIPJ flexed over the edge of a table (and will have hyperextension of the DIPJ).**
- Apply splint to hold the PIPJ straight and refer to the next fracture clinic.

Boutonniere deformity



10. SWAN NECK DEFORMITY

- Swan neck deformity is a deformity of the digits that consists of:
 - Hyperextension of the proximal interphalangeal (PIP) joints
 - Compensatory flexion of the distal interphalangeal (DIP) joints

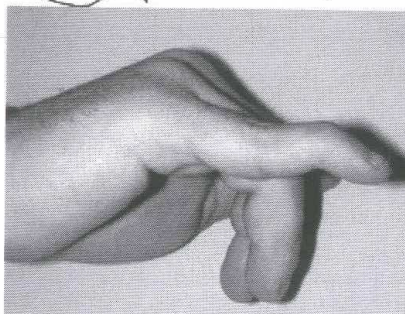
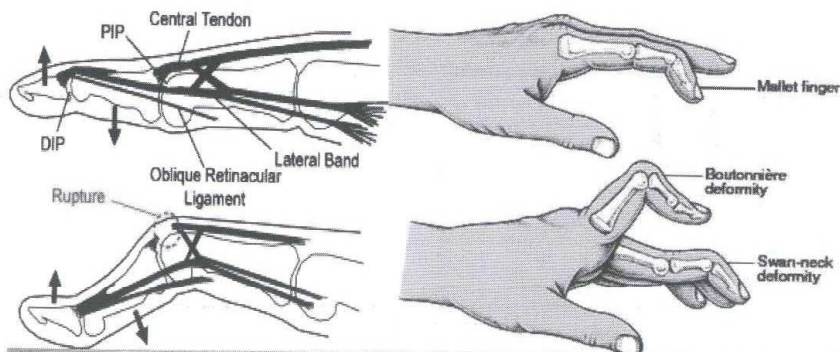


Figure 4.8.32. Swan-neck deformity

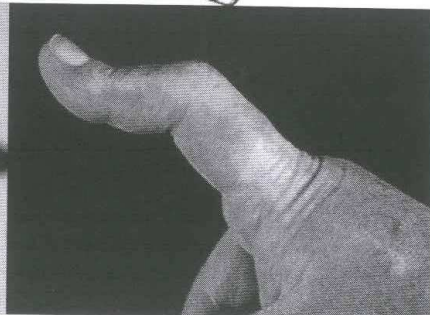


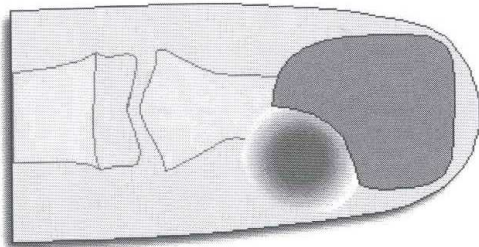
Figure 4.8.33. Boutonniere deformity

11. SUBUNGUAL HAEMATOMA

- Crush injury
- X-Ray for fracture if significant force / pain
- Trephine & drain haematoma if > 25% nail
- Nail removal not indicated
- Non-adherent dressing
- GP follow up
- No antibiotics for uncomplicated subungual haematoma
- Antibiotics if fracture and wound open / intervention

12. PARONYCHIA / FELON FRACTURE

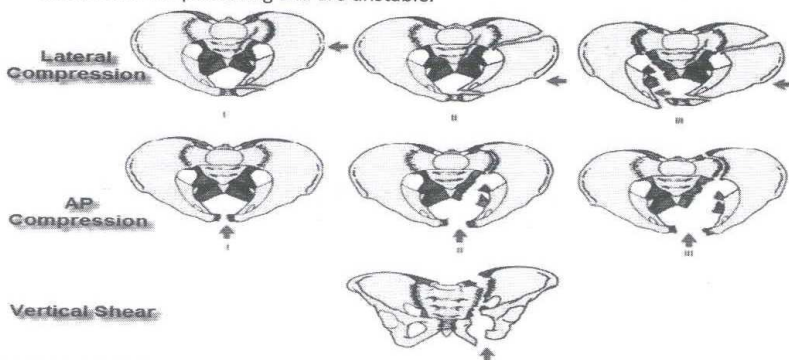
- Acute paronychia develops over a few hours when a nail fold becomes painful, red and swollen.
- Throbbing pain indicated presence of pus



- Usually staphylococcus
- F: M = 3:1
- Ask about background **Diabetes, Immunosuppression, Raynaud's or fungal** infections
- Acute case treat with incision / elevation nail fold (image right) rather than antibiotics
- Recurrent paronychia or if nail bed involvement best treated by removing whole nail
- **Paronychia DDx includes**
 - Felon
 - Herpetic whitlow
 - (right) Malignant tumours
 - Fungal infection
 - Pemphigus vulgaris²³
 - Splinters, foreign body
- **Felon**
 - o Deeper pulp space infection
 - o Risk of osteomyelitis of distal phalanx
 - o Treat with I&D - longitudinal incision parallel with nail

VII. PELVIC TRAUMA

- **Pelvic fractures** can be simple or complex and can involve any part of the **bony pelvis**.
- Pelvic fractures can be fatal, and an unstable pelvis requires immediate management.
- **EPIDEMIOLOGY**
 - Pelvic fractures can be seen in any group of patients.
 - Like much trauma, there is a bimodal distribution with younger male patients involved in high-energy trauma and older female patients presenting after minor trauma.
- **CLINICAL PRESENTATION**
 - Patients tend to present following trauma with pelvic/hip pain.
 - They will often be immobilised by ambulance crews on arrival and potentially have other life-threatening conditions associated with high-energy trauma.
- **PATHOLOGY**
 - Most pelvic fractures result from trauma:
 - Motor vehicle collision (~50%)
 - Pedestrian vs. Motor vehicle (~30%)
 - Fall from height (~10%)
 - Motorbike collisions (~4%)
 - Other e.g. Sports injury, low-energy fall
 - Pelvic insufficiency fractures are common in the elderly.
 - The type of fracture that occurs is a result of the type of injury (impact or compression), the energy involved and the strength of the bones.
 - The potential morbidity associated with these fractures is related to the involvement of the pelvic ring. Injuries that result in disruption of the pelvic rings result in a significantly worse prognosis. Direct impact low-to-moderate energy injuries usually result in a solitary and localised fracture. Compression injuries tend to cause fractures that involve the pelvic ring and are unstable.



- **CLASSIFICATION**
 - Four main forces have been described in high-energy blunt force trauma that results in unstable pelvic fractures:
 - **Anteroposterior compression:** result in an **open book** or **sprung pelvis** fractures
 - **Lateral compression:** result in a **windswept pelvis**
 - **Vertical shear:** results in **bucket handle** fracture
 - **Combined mechanical:** occur when two different force vectors are involved and results in a complex fracture pattern

- Isolated stable pelvic fractures can also occur in the context of lower energy mechanisms or sporting injuries:
 - Acetabular fracture
 - Pubic ramus fracture
 - Iliac wing fracture (**Duverney fracture**)
 - Avulsion fractures (e.g. **ASIS**, iliac crest, ischial tuberosity)
- **ASSOCIATED INJURIES**
 - Pelvic fractures carry a significant risk of uncontrolled pelvic bleeding and exsanguination from pelvic fractures is a real possibility.
 - This may result in pelvic, thigh and/or **retroperitoneal haemorrhage**.
 - Pelvic angio-embolisation should be considered in patients with evidence of persistent blood loss with no evidence of intra-abdominal bleeding prior to surgical fixation.
- **OTHER COMPLICATIONS INCLUDE:**
 - *Bladder rupture*
 - *Urethral rupture*
- **RADIOGRAPHIC FEATURES**
 - The radiographic features are varied and even for serious and severe injuries can be subtle on plain radiographs.
 - **X-ray**
 - X-ray is a quick and simple test that will detect the majority of pelvic fractures.
 - They can be difficult to assess because of the complexity of the shape of the sacrum, pelvis and proximal femora.
 - **CT**
 - CT is the modality of choice for accurately depicting complex acetabular or pelvic ring fractures.
 - After an initial plain radiograph, a CT is often required to make an accurate assessment of the fracture.
- **TREATMENT AND PROGNOSIS**
 - Treatment and prognosis depend on the type of injury:
 - **Simple ramal fractures** are treated by immobilisation.
 - **Multi-part acetabular fractures** require reconstruction by an experienced operator.
 - **Complex pelvic ring fractures** may require external fixation. In these patients, their prognosis is partly dependent on their comorbidities and other related injuries.
 - Pelvic fractures carry a significant mortality and morbidity.
 - It has been reported that ~75% of pre-hospital deaths from motor vehicle collisions are secondary to pelvic fractures.
- **ED MANAGEMENT OF PELVIC INJURIES**
 - Generic trauma management principles apply.
 - Assessment of the pelvis occurs during 'C' assessment in the primary survey.
 - **A pelvic X-ray** is part of the trauma series of X-rays and should be performed as an adjunct to the primary survey.
 - Patients should be X-rayed in the resuscitation room to identify fractures.
 - Manipulation of the pelvis to determine instability is not recommended.
 - The log roll should be delayed until the pelvis has been 'cleared'.
 - Excessive movement may disrupt clots that have formed.

- If a pelvic fracture is identified, the patient should be **scooped** for transfer and not log rolled. **Pelvic splinting** may be used for unstable fractures to close the increased volume of the pelvis.

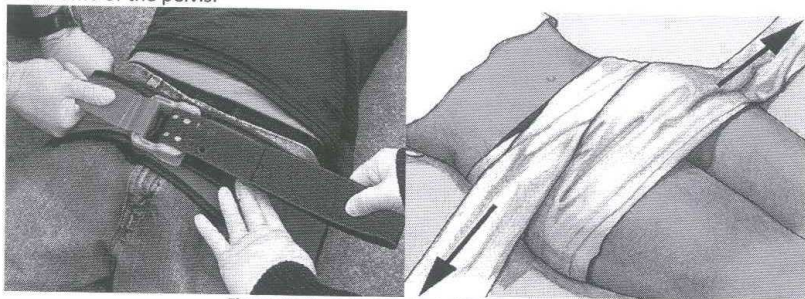


Figure 4.8.34. Pelvic Binder and Pelvic sheet

- If no splint is available, a **sheet** can be used as a temporary holding measure. The sheet should be wrapped around the pelvis at the level of the anterior superior iliac spines and the legs internally rotated and secured in this position. The aim is to reduce the volume of the pelvis and allow tamponade of bleeding points.
- Patients should be **referred early to the trauma surgeons**.
- If the patient is haemodynamically unstable, a **FAST** should be performed to determine the need for laparotomy as well as pelvic fixation.
- Pelvic injuries may be managed operatively (internal or external fixation/ packing) or by interventional radiology (angiography /embolization).

1. SACRAL FRACTURES

- Sacral fractures can be difficult to identify on X-ray, alignment of the sacral alar and the sacro-iliac joints should be carefully assessed.
- **Sacral nerve roots** may be damaged, so check **bladder and bowel function, saddle sensation, and lower limb function**.

2. ACETABULAR FRACTURES

- Usually associated with traumatic posterior hip dislocation (e.g. from knees hitting the dashboard in a RTC).
- In major trauma, resuscitate and prioritize other injuries, as required.
- Longitudinal traction of the lower extremity may be useful.
- Massive haemorrhage may be a problem.
- Risk of damage to the **sciatic nerve**.
- Later problems with **arthritis** are likely.
- Once the patient is stable, **dedicated Judet views (45° oblique)** or **CT of acetabulum** will help guide operative management.

3. COCCYGEAL FRACTURES

- May result from a heavy fall on to the bottom.
- Check for **rectal tears/damage** (refer if present).
- X-ray is rarely required, diagnosis is clinical.
- Majority are managed conservatively with advice (sit on ring cushion) and analgesia.
- If grossly displaced, may require manipulation (with anaesthetic) by orthopaedic team.

4. NECK OF FEMUR FRACTURE**• BACKGROUND**

- Stress fractures of the femoral neck are uncommon injuries.
- In general, these injuries occur in 2 distinct populations:
 - **Young**, active individuals with unaccustomed strenuous activity or changes in activity, such as **runners or endurance athletes**
 - **Elderly** individuals with **osteoporosis**.
- Elderly individuals may also sustain femoral neck stress fractures; however, hip fractures are much more common and are often devastating injuries.

• Causes of NOF Fracture:

- **High-energy trauma**: in young patients, are often associated with multiple injuries and high rates of avascular necrosis and nonunion.
- **Osteoporosis**
- **Metastases**
- **Pagets disease**
- **Osteomalacea**
- **Hyperparathyroidism**

Risk factors

- The risk factors for a fractured NOF can simplistically be divided into risk factors for falls and risk factors for osteoporosis (though there are some things eg alcohol, immobility which may be risk factors for both)

RISK FACTORS FOR OSTEOPOROSIS		RISK FACTORS FOR FALLS
Age	Age ≥ 75 years	Gait deficit.
Inactivity	History of previous falls.	Balance deficit
Current smoking	Fear	Visual impairment.
Excessive alcohol intake	Acute illness	Mobility impairment
BMI <18.5	Neuromuscular disorders	Cognitive impairment.
Heredity.	Multiple medications	Decreased hearing,
	Home hazards.	Urinary incontinence.
		Living alone

- A patient who has had one osteoporotic fracture doubles their risk of a further fracture

CLINICAL ASSESSMENT**• Diagnosis of the cause of the fall**

- As well as diagnosing the fracture, it is also important to diagnose the cause of the fall as there may be a treatable cause eg postural hypotension, cardiac arrhythmia.
- What are often described as mechanical falls (eg trips and slips) may have multifactorial causes as described above.
- Even if there is no medical cause for the fall, it is important to identify co-morbidities as these may influence patient management, even in the ED.
- There is not always a history of a fall. Sometimes a fall cannot be remembered (eg as a result of dementia) or described (eg due to dysphasia) and some pathological fractures may occur spontaneously.

• Clinical features

- The classical deformity is that the leg is **shortened and externally rotated** but 15% of fractures are impacted and there will be no deformity.

DIAGNOSIS OF THE TYPE OF FRACTURE

- It is important to diagnose the type of fracture as this influences treatment. The usual classification describes the fracture in relation to the joint capsule:
 - Intracapsular fractures**
 - Extracapsular fractures**
- The importance of this classification is that a large proportion of the blood supply to the head of the femur comes via the capsule, so in intracapsular fractures, there is a risk of **non-union and avascular necrosis**.
- For this reason, **arthroplasty** (either hemiarthroplasty or total hip replacement) may be indicated for these fractures.

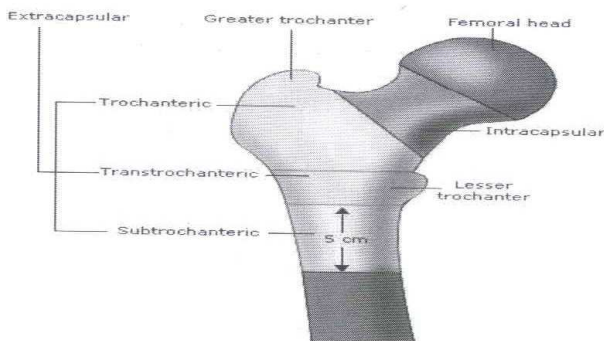
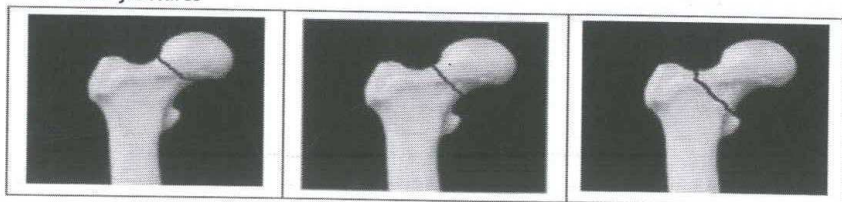


Figure 4.8.35. Anatomy of the proximal femur

- Intracapsular fractures** can be further divided according to their site into:
 - Subcapital fractures**
 - Transcervical fractures**
 - Basal fractures**



Subcapital fracture

Transcervical fracture

Basal fracture



- Basal fractures are, technically, intracapsular injuries but behave like extracapsular injuries and for prognosis purposes are usefully classified as such.

GARDEN CLASSIFICATION OF INTRACAPSULAR FRACTURE

- o **Type 1:** Impacted fracture or abduction fraction
- o **Type 2:** Undisplaced
- o **Type 3:** Some displacement
- o **Type 4:** Grossly displaced



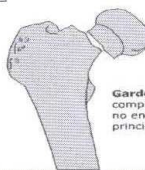
Garden I fracture
incomplete and minimally displaced.



Garden II fracture
complete, nondisplaced.



Garden III fracture
complete fracture and partially displaced.



Garden IV fracture
completely displaced, with no engagement of the 2 principal fragments.



Undisplaced intracapsular fracture



Displaced intracapsular fracture

- **Extracapsular fractures include:**
 - o *Trochanteric fractures*
 - o *Transtrochanteric fractures*
 - o *Subtrochanteric fractures*

DIFFERENTIAL DIAGNOSIS

- An elderly patient with pain in the hip following a fall must be assumed to have a fractured neck of femur until proved otherwise.
- Other fractures that can occur with this mechanism are:
 - o **Fractures of the pubic ramus**
 - o **Fractures of the acetabulum.**

INVESTIGATION STRATEGIES

- In addition to appropriate imaging, the BOA also recommends that the following investigations are done in the ED:
 - o **FBC, U&E, Group and save**
 - o **ECG**
 - o **Pre-operative chest X-ray** (except in younger fitter individuals)

- Have a very low threshold for X-raying to exclude a fracture.
- Any elderly patient who falls should have an X-ray of the hip if they complain of pain anywhere between the waist or knee. Any elderly person who complains of hip pain or goes off legs, even without a history of a fall, should also have an X-ray of the hip.
- **X-rays**
- The usual X-rays obtained for a patient with a hip injury are:
 - **AP pelvis.** This shows both hips for comparison and will also show other fractures (eg pubic rami) that may also occur in a fall.
 - **Lateral of the hip.** This is essential as not all fractures will show on an AP X-ray
 - Fractures are commonly missed.
 - To avoid missing fractures, look for:
 - **Shentons Line:** This is an imaginary line drawn up the inferior neck of the femur and extending along the inferior part of the superior pubic ramus. This should be smooth. If it is disrupted, this may indicate a fracture.
 - Follow the trabeculae along the neck of the femur and ensure that they are intact.
 - A sclerotic line across the neck of the femur may indicate an impacted fracture
 - Look for steps in the cortex.



Shentons line

Extracapsular fracture

- If the X-ray is thought to be normal, there are two possibilities:
 - Most likely is that the X-ray is normal. However, fractures are not always easy to see and it is possible that there is a fracture that has been missed. If the clinical features suggest a fracture but the X-ray appears normal, it is important that the X-ray is re-examined and consider obtaining a second opinion.
 - In the majority of cases, a normal X-ray means that there is no bony injury but about 1% of hip fractures are not visible on initial X-rays. There are no validated guidelines on the management of patients with hip pain following a fall but with no obvious fracture visible on an X-ray.
 - *A pragmatic approach to the problem would be:*
 - Give analgesia and try to mobilise the patient.
 - If the patient walks well, there is no fracture and they can be discharged.
 - If the patient cannot walk they will need admission
 - Try again to mobilise the patient the following day: if they are still immobile, they will need further imaging to exclude an occult fracture.
 - The best form of imaging to exclude a fracture is an MRI. If it is not possible to obtain an MRI, **CT scan** or a **bone scan** may be performed or further plain films obtained after 48 hours.

ED MANAGEMENT OF NOF**a. Management of pain**

- o The best method of giving systemic analgesia is to **titrate doses of opiate intravenously**.
- o **Entonox** may also be useful.
- o The most effective form of local analgesia is a local anaesthetic block (eg. **fascio-iliaca or femoral nerve block**).
- o This should be done before a potentially procedure eg moving a patient in the X-ray department or moving a patient to a bed.
- o **NSAIDs** should be avoided because of the risk of gastrointestinal complications and their effect on renal function in the elderly.

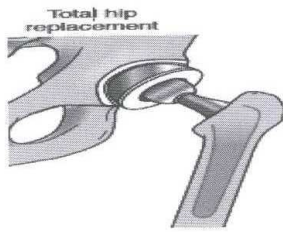
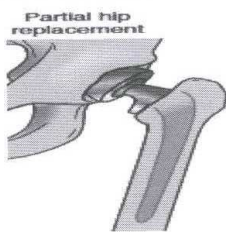
b. Refer to ortho:

- o Rapid transfer to an orthopaedic ward is important. Patients must be transferred to a ward **within 4 hours** to avoid pressure sores, confusion and pain.
- o The CEM additionally recommends that X-rays should be performed within 60 minutes and that 90% of patients should be admitted within 2 hours.
- o Although it is important for patients to be transferred quickly to an orthopaedic ward, the majority of patients with this fracture have co-morbidity and they may have fluid balance problems because of diuretics, renal impairment etc.
- o They should have input from an orthogeriatrician, and consideration should be given to formal shared care.

PROGNOSIS & FOLLOWUP STRATEGIES

- While the prognosis following a hip fracture is very dependent on the degree of frailty before the injury and the pre-existing medical problems, there are some statistics that may act as a guide:
 - o **The 30 day in hospital mortality is about 10%.**
 - o **The 12-month mortality is about 30%**
 - o **Risk of DVT up to 91%.**
 - o **Risk of pulmonary embolus (PE): is 10-14%** (but the incidence of clinically apparent PE is only about 1% and the incidence of death due to PE is about 0.5%).
 - o Other complications include **haematoma formation, superficial and deep infection, loosening of prostheses and peri-prosthetic fracture.**
 - o **Pressure sores** are common but should be avoidable

SURGICAL TREATMENT	INDICATION
Internal fixation	Physiologically young and active patients < 60years
Total hip replacement	Active, independent patients > 60years
Hemiarthroplasty	Elderly patients with functional limitations and dependent living status



5. FEMUR FRACTURE

PREHOSPITAL CARE

- Treatment of a patient who complains of hip pain should include immobilization on a stretcher. If the patient is a victim of multiple traumas, **address the ABCs and immobilize the cervical spine as appropriate.**
- If fracture or deformity of the femur is obvious, **apply a traction splint and place an intravenous (IV) line for hydration.**
- If the patient is hypotensive or tachycardic, initiate **crystalloid fluid bolus and place patient on supplemental oxygen.**

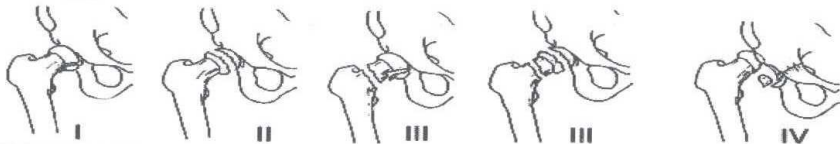
EMERGENCY DEPARTMENT CARE

- Attend to the ABCs first and conduct a thorough search for other possible injuries.
- In cases of obvious femur fracture:
 - Immobilize the patient,
 - Place 2 large-bore IV lines for hydrations and possible transfusion,
 - Restrict the patient's oral intake to nothing by mouth (NPO),
 - Obtain specimens for preoperative labs if necessary. Orthopedic treatment decisions vary significantly among different practitioners, thus early consultation for all hip fractures is recommended.
 - Initiate appropriate parenteral analgesia as soon as possible.
- Ultrasound-guided **Femoral nerve blocks** or **Fascia Iliaca Compartment Block (FICB)** may also be used to achieve adequate analgesia.
- **A muscle relaxant** also may be necessary.
- Administer **antibiotics to cover skin flora** (ie, cefazolin sodium)
- **Tetanus immunization**, as necessary, in open fractures.

A. FEMORAL HEAD FRACTURES

PIPKIN CLASSIFICATION

- It is the most commonly used classification for **femoral head fractures**, which are uncommon but are associated with **hip dislocations**.
 - **Type I:** fracture distal to the fovea capitis
 - **Type II:** fracture proximal to the fovea capitis
 - **Type III:** type I or II fracture with a fracture of the femoral neck, has an increased risk of **avascular necrosis**
 - **Type IV:** type I or II fracture with a fracture of the acetabular wall, usually the posterior wall



MANAGEMENT

- For **type 1 femoral head fractures**, orthopedic consultation in the ED should be obtained.
- Treatment is to reduce dislocated femoral head and fracture fragment as soon as possible to avoid avascular necrosis.
- Small fracture fragments may need to be removed. If a single attempt at closed reduction fails, then open reduction and internal fixation (ORIF) is the next treatment of choice.
- For **type 2**, early orthopedic consultation for admission and arthroplasty is recommended.

VIII. KNEE / LEG INJURIES

- **History**

- Nature of injury, e.g. direct, indirect, rotational, presence of locking, giving way.
- Previous knee problems.

- **Examination**

- **Look:** wasting, deformity, swelling, bruising.
- **Feel:** effusion, localise tenderness, check stability, popliteal swelling?
- **Move:** check range, exclude locking by comparing extension on both sides
- **Special tests:** McMurray's test.

- **X-ray**

- **AP & Lateral views:** see Ottawa Knee rules below.
- Additional views should include **skyline view** for patellar problems and **tunnel view** for intercondylar area (e.g. loose bodies)

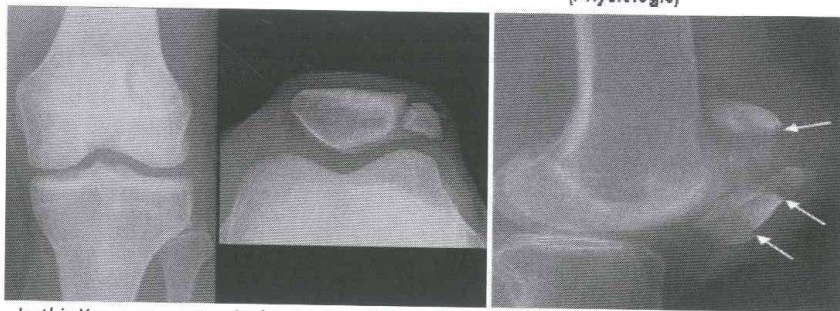
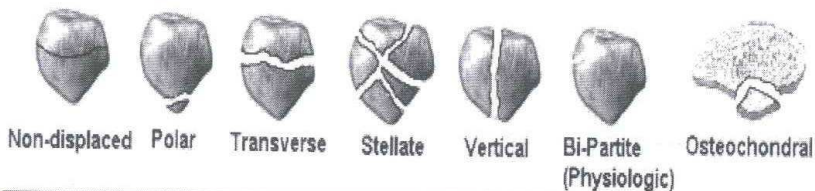
OTTAWA KNEE RULE

- A Knee X-ray series is only required for knee injury patients with *any* of these findings:
 - Age ≥ 55
 - Isolated tenderness of the patella (no bone tenderness of the knee other than the patella).
 - Tenderness at the head of the fibula.
 - Inability to flex to 90° .
 - Inability to bear weight both immediately and in the ED (4 steps; unable to transfer weight twice onto each lower limb regardless of limping).
- **Aspiration**
 - Evidence for aspiration to improve symptoms?
 - Indications are:
 - Tense haemarthrosis and
 - Diagnostic uncertainty

1. PATELLAR FRACTURES

- Patella fracture is one of the common knee injuries usually post direct trauma to the patella (fall on the knee or dashboard) or sudden forceful contraction of the quadriceps muscles in a context of sport injury.
- **Clinical presentation**
 - Patients present with marked swelling and pain over the patella with point tenderness and marked reduction in extension strength.
 - Usually there is a large joint effusion or hemarthrosis.
- **Mechanism of injury**
 - There are different causes for patella fracture:
 - Direct blow to patella, e.g. Dashboard injury
 - Severe forces by extensor mechanism
 - After anterior cruciate ligament reconstruction
 - After total knee reconstruction
 - Pathological fracture
- **Morphology**
 - Transverse fracture in mid-patella (most common)

- o Comminuted fracture
- o Vertical fracture (rare)
- o Osteochondral defect usually from medial facet
- o Patellar sleeve fracture in children
- **Differential diagnosis**
 - o **Bipartite patella:** well corticated



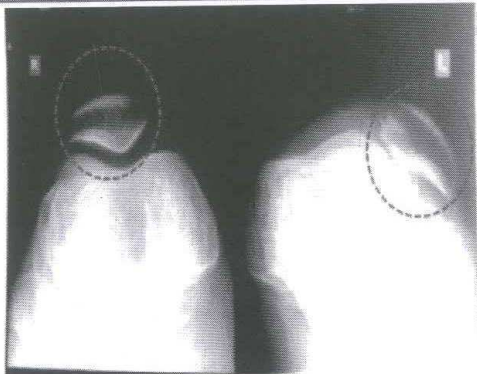
In this X-ray, an anatomical variation of the patella can be seen – the bipartite patella, in which the patella is split into two parts.

This x-ray of the knee taken from the side shows a patella that has been fractured in three places.

- **MANAGEMENT IN ED**
 - o **If undisplaced:** apply POP backslab and refer to the Fracture Clinic
 - o **If displaced:** refer to the on-call Orthopaedic Team.
 - o Most need internal fixation as quads tone distracts the fragments
 - o For the treatment of transverse fractures, the classic method is the tension band.

2. DISLOCATED PATELLA

- **Lateral patellar dislocation** refers to lateral displacement followed by dislocation of patella due to disruptive changes to the medial patellar retinaculum.
- **Epidemiology**
 - o Patellar dislocation accounts for ~3% of all knee injuries and is commonly seen in those individuals who participate in sport activities.
- **Mechanism**
 - o Patella dislocation most commonly results from a twisting motion, with the knee in flexion and the femur rotating internally on a fixed foot (valgus-flexion-external rotation).
- **RADIOGRAPHIC FEATURES**
- **Plain radiograph**
 - o Lateral displacement of patella noted on **skyline projection**
 - o Joint effusion



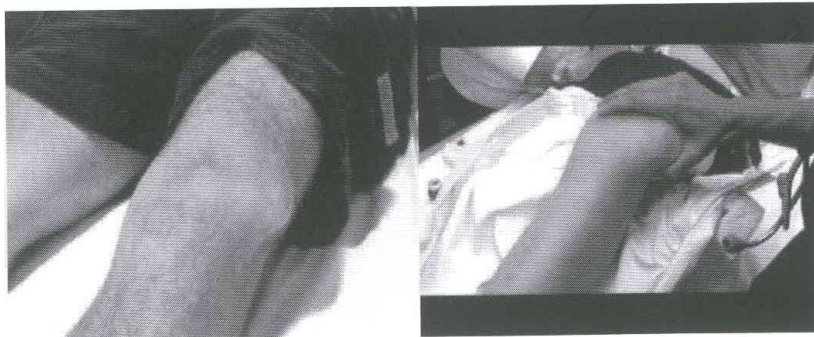
- Left patella dislocated laterally out of the trochlear notch
- Left trochlear sulcus shallower than right.

• **MRI**

- The following features are noted:
 - Medial retinacular abnormalities (ranging from strain to complete disruption) with adjacent periligamentous edema and haemorrhage.
 - Lateral displacement of patella.
 - Medial patellar contusion +/- corresponding lateral femoral condyle contusion.
 - Joint effusion.
 - The presence of an abnormal medial retinaculum should suggest the diagnosis of transient lateral patellar dislocation.

• **DIFFERENTIAL DIAGNOSIS**

- **Acute ACL tear:** no medial patella contusion in this injury
- **Direct trauma to lateral knee:** normally no patellar contusion



• **MANAGEMENT:**

- Usually reduced before presentation - if not, reduce by extending hip (Entonox)
- X-ray to exclude fracture
- If first episode, treat conservatively in preference to surgery
- If first episode, apply cricket bat splint in preference to POP cylinder
- Refer to the Fracture Clinic
- Consider prophylactic anticoagulation (LMWH) if high risk of VTE or prior DVT.

3. KNEE DISLOCATION

• RELEVANT ANATOMY

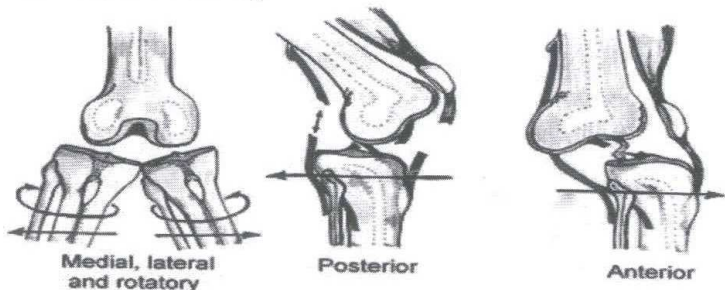
- The femoral artery gives rise to the popliteal artery. The collateral blood supply system of the lower leg breaks off from the popliteal artery below the knee joint.
- The popliteal artery is **held down firmly at either end of the knee joint**, proximally by the tendinous hiatus of the adductor magnus muscle and distally by the tendinous arch of the soleus muscle. As a result, the popliteal artery is **anchored down like a bowstring**, placing it at risk for injury during knee dislocations.

• IMAGING

- **Plain X-rays:** Should be obtained if patient has good peripheral pulses. Used to confirm the clinical findings and document associated fractures (femur and tibial).
 - Check for asymmetric / irregular joint space
 - Check for avulsion fxs
 - **Second sign** – lateral tibial condyle avulsion fracture.
- **Standard angiography:** Standard of care modality.
- **CT angiography:** Provides accurate non-invasive assessment of vascular injury and is most commonly used in place of standard angiography.
- **Color flow Doppler ultrasonography:** Gaining acceptance as an alternative to standard angiography in select groups of low-risk patients.
- **MRI:** Helps determine extent of injury, will identify ligamentous injury, joint capsule, meniscus, and articular cartilage integrity. Rarely used in acute management of knee dislocations.

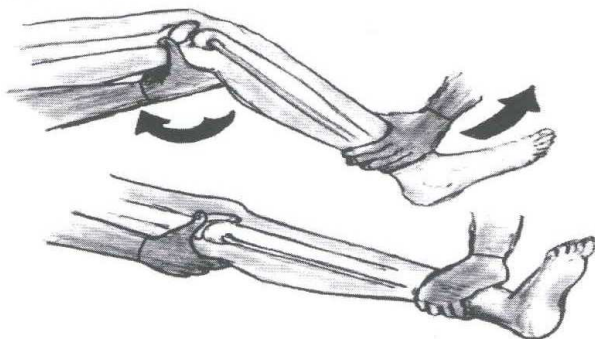
• CLASSIFICATION

- Classification is based on the position that the **tibia is displaced relative to the femur**.
- There are five general types of knee dislocations in order of frequency:
 - **Anterior: most common** dislocation (50-60%) and occurs from hyperextension of the knee resulting in tearing of the posterior structures. This injury drives the distal femur posterior to the proximal tibia.
 - **Posterior: most commonly associated with popliteal artery injury.** Usually results from direct blow to the proximal tibia displacing it posterior to the distal femur.
 - **Lateral or Medial**
 - **Rotatory:** There are 2 types of rotator dislocations with posterolateral being the most important, although rare. Posterolateral dislocations cannot be reduced by closed reduction. These results from the body rotating in opposite direction of the remainder of the lower leg.



• MANAGEMENT

- The first step in management involves **immediate reduction of obviously dislocated knee, especially if neurovascular compromise exists, without radiographs.**
- **Neurovascular status** should be documented **before and after attempting reduction.**
- Prior to reduction evaluate the patient for signs of a posterolateral dislocation (ie "**Dimple sign**"), as these dislocations are not amenable to closed reduction.
- An anteromedial skin furrow, or "**dimple sign**" at the medial joint line, is suggestive of a posterolateral dislocation, which are **irreducible.** Attempts at closed reduction may compromise the thin veil of skin overlying the prominent femoral condyle in posterolateral dislocations leading to **skin necrosis.**
- The initial approach to reducing all knee dislocations is to **apply longitudinal traction** to the extremity. This is usually all that is required to reduce a knee.
- Anterior knee dislocations may require additional lifting of the distal femur, whereas posterior dislocations may require lifting of proximal tibia to complete reduction.
- After reduction, the knee should be **immobilized in a long leg posterior splint with the knee in 15-20 degrees of flexion.**



• COMPLICATIONS

- **Short-term complications:**
 - Popliteal artery injury
 - Compartment syndrome of leg
 - Common peroneal nerve injury (**Foot Drop**)
 - Associated fracture or ligamentous injury
 - DVT
- **Long-term complications:**
 - Pseudoaneurysm
 - Early osteoarthritis
 - Stiffness
 - Chronic pain

• CLINICAL PEARLS

- Assume all patients with suspected knee dislocation have **vascular injury until proven otherwise.**
- **Nearly half of all knee dislocations spontaneously reduce prior to ED presentation.**
- **Posterior** knee dislocations are more commonly associated with popliteal artery injury.
- A normal physical examination alone **does not reliably exclude vascular injury.**

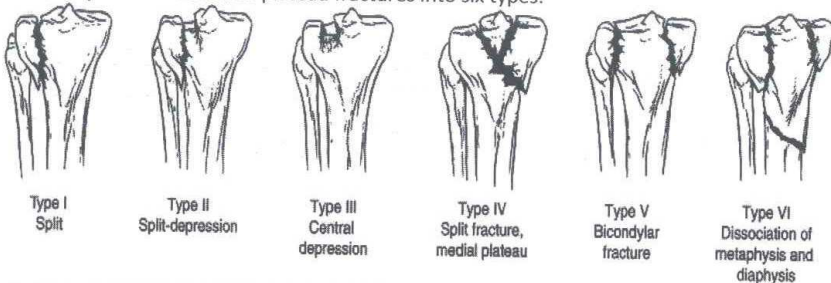
4. FRACTURES OF THE TIBIAL SHAFT

- Beware compartment syndrome in all (even apparently simple fractures).
- Record neurovascular status.
- Analgesia, above knee POP and Refer to the on-call Orthopaedic Team

5. TIBIAL PLATEAU FRACTURE

SCHATZKER CLASSIFICATION

- This system divides tibial plateau fractures into six types:



6. FRACTURES SHAFT OF FIBULA

- Ensure no ankle diastasis by requesting ankle X-rays.
- Check **common peroneal nerve**.
- If no other abnormality bandage or plaster for comfort and refer to the Fracture Clinic.

7. ANTERIOR CRUCIATE LIGAMENT INJURY

- **Pathology**
 - The ACL is the most commonly disrupted ligament of the knee, especially in athletes who participate in sports that involve rapid starting, stopping, and pivoting (e.g. soccer, basketball, tennis, netball and snow skiing).
- **Associations**
 - **O'Donoghue's unhappy triad**
- **Radiographic features**
 - An avulsion of the tibial attachment may be seen in younger patients.
- **Radiograph**
 - Deep lateral sulcus sign
 - Anterior tibial translocation sign
 - Segond fracture
 - Arcuate fracture
 - Joint effusion
- **CT arthrography**
 - Considered to have high specificity and sensitivity in detecting ACL disruption.
 - CT is helpful in characterising the avulsion bone fragment, when it is present.
- **MRI**
 - Imaging of ACL tears should be divided into primary and secondary signs.
 - **Primary signs** are those that pertain to the ligament itself.
 - **Secondary signs** are those which are closely related to ACL injuries.

8. LARGE EFFUSION OR HAEMARTHROSIS

- Aspirate under full aseptic conditions, unless immediate referral or surgery intended because of severity of injury.
- If frank blood in the joint:
 - Immobilise in POP knee backslab and refer to Fracture Clinic
 - Refer to on-call Orthopaedic Team
 - There is no evidence to support early MRI in acute traumatic knee haemarthroses with normal x-rays

9. LOCKED KNEE

- The knee will flex but will not fully extend.
- Refer to the on-call Orthopaedic Team.

10. COLLATERAL LIGAMENT TEAR

- Tenderness and pain on stressing the ligament.
- If there is definite laxity and marked bruising/swelling obtain an opinion from consultant.
- Complete ligament rupture can be masked by muscle spasm.
- If little laxity or pain is evident, apply tubigrip and refer to the physiotherapist.

11. POSSIBLE MENISCAL TEAR

- If **not** locked, apply a double tubigrip and provide crutches.
- Refer ED physio clinic.
- **Possible torn cruciate ligaments.**
- Rest, crutches, physiotherapy referral and GP follow up.
- ACL rupture patients are NOT routinely referred to the fracture clinic or orthopaedic follow up as conservative management gives similar functional results to (early or late) surgery.
- These patients should NOT be referred to the ED clinic.

12. SECOND FRACTURE

- It is an **avulsion fracture of the knee** that involves the lateral aspect of the tibial plateau and is very frequently (~75% of cases) associated with disruption of the **anterior cruciate ligament (ACL) tear**.



IX. ANKLE & FOOT INJURIES

HISTORY

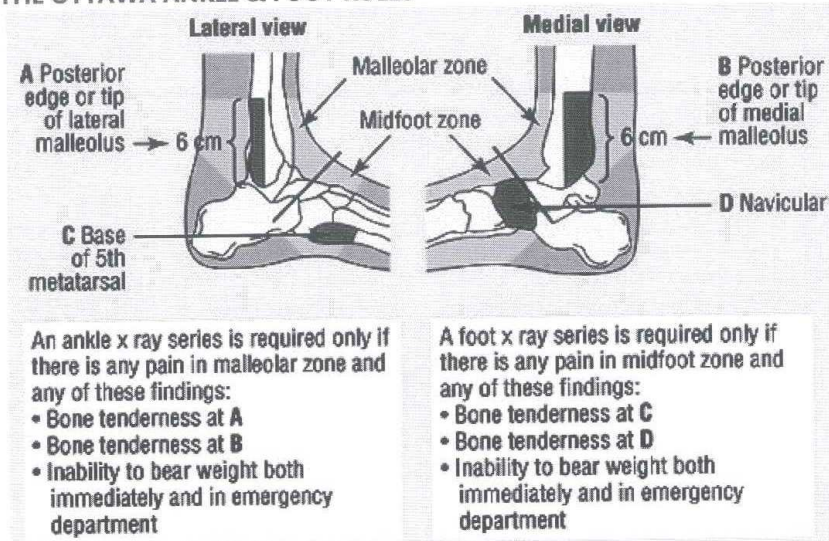
- The mechanism of injury is of vital importance and may give valuable clues towards the diagnosis of injuries to the feet and other associated injuries. Examples are:
 - **An inversion injury of the ankle** may cause an ankle injury but may also cause a fracture at the base of the 5th metatarsal.
 - **A fall from a height onto the heel** typically causes a fractured calcaneum. Patients may land on both heels so bilateral fractured calcaneum is not uncommon and the force that causes a fracture of the calcaneum may also cause a fracture of the lumbar spine. If there is any suspicion of back injury (or if a back injury cannot be ruled out because of distracting pain) the lumbar and thoracic spine must also be X-rayed.
 - **Dropping a weight onto the foot** may cause a fracture but is unlikely to cause a dislocation whereas a stubbed toe may cause either a fracture or a dislocation.
- As with any other injury, note any past medical history that might influence the presentation or management of a foot injury e.g.
 - Diabetic neuropathy may predispose to a neuropathic arthropathy
 - Previous foot injuries or congenital foot deformity
 - Conditions that might affect giving an anaesthetic

EXAMINATION

- Examination will consist of:
 - **Look** – for deformity, bruising, swelling scars etc
 - **Feel** – for deformity, point of maximum tenderness
 - **Move** – examine for movement at all the joints – subtalar, midtarsal, toes
 - **Function** – can the patient walk? Examine their gait
 - **Test for neurovascular function**
 - **Examine the ankle**

INVESTIGATION STRATEGIES

- **X-ray**
 - As noted above, inversion of the ankle may be associated with a foot injury.
 - Give evidence-based advice on whom to X-ray.
 - These state that in ankle injuries, foot X-rays are only required if:
- A “standard” foot series of X-rays consist of an **AP, an oblique and a lateral of the foot**.
- Suspected talar injuries will require ankle X-rays in addition to foot X-rays
- The lateral X-ray is most useful for looking at the hind foot and mid foot and these areas are also well shown on a lateral ankle x-ray so if an ankle X-ray series has been obtained, a lateral foot x-ray will rarely be required.
- Fractures (and other abnormalities) always show best if the X-ray is centred over the abnormality so, for a toe injury, request **X-rays of the toes** rather than a foot X-ray.
- Calcaneal fractures may not be easy to see on ankle or foot X-rays so if a fracture is suspected, request **calcaneal views** (a lateral view of the calcaneum or ankle and an axial view of the calcaneum). Even if you forget to request specific views, if you state the injury you suspect or want to exclude, the radiographer should do the X-rays that you need.
- **Computed tomography (CT)**
 - CT will often be needed to assess the extent of calcaneal and talar fractures and also mid-tarsal and tarso-metatarsal injuries.
 - CT may also be useful if the diagnosis is not obvious.

THE OTTAWA ANKLE & FOOT RULES**SPECIFIC MANAGEMENT IN THE ED****1. ANKLE FRACTURES****DIAGNOSIS****• History**

- Ankle fractures are usually due to a twisting mechanism sustained as a result of a low-energy injury.
- The position of the ankle at the time of injury
- A higher energy mechanism: the pilon fracture.
- Medical comorbidities such as diabetes, peripheral vascular disease and smoking, which can complicate wound and fracture healing.
- A social history should be taken to identify the patient's pre-injury level of mobility, home situation and regular activities as well as their future functional aspirations.

• Examination

- Look, feel and Move
- The neurovascular status of the limb should be checked before and after reduction.

• RADIOLOGICAL FEATURES

- A standard radiological series of the ankle, including anteroposterior, lateral, and mortise radiographs, is generally sufficient to classify these injuries and plan treatment.
- Where a patient has more proximal leg tenderness or medial clear space widening with no obvious fibular fracture, full-length radiographs of the tibia and fibula should be obtained to rule out the presence of a Maisonneuve injury.
- More complex axial imaging is rarely indicated; exceptions include triplane and pilon fractures.

WEBER CLASSIFICATION ANKLE FRACTURES

- o **Type A** - Infrasyndesmotic
- o **Type B** - Transsyndesmotic
- o **Type C** - Suprasyndesmotic

Weber type

Weber A

- Occurs below the syndesmosis, which is intact.
- According to **Lauge-Hansen**, it is the result of an adduction force on the supinated foot.
- **Stage 1** - Tension on the lateral collateral ligaments results in rupture of the ligaments or avulsion of the lateral malleolus below the syndesmosis.
- **Stage 2** - Oblique fracture of the medial malleolus.

Weber B

- This is a transsyndesmotic fracture with usually partial - and less commonly, total - rupture of the syndesmosis. According to Lauge-Hansen, it is the result of an exorotation force on the supinated foot.
- **Stage 1** - Rupture of the anterior syndesmosis
- **Stage 2** - Oblique fracture of the fibula (this is the true Weber B fracture)
- **Stage 3** - Rupture of the posterior syndesmosis or - fracture of the malleolus tertius
- **Stage 4** - Avulsion of the medial malleolus or - rupture of the medial collateral bands

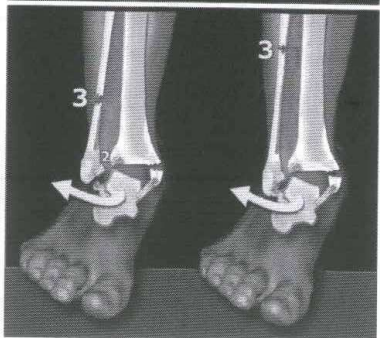
Weber C

- This is a fracture above the level of the syndesmosis. Usually there is a total rupture of the syndesmosis with instability of the ankle. According to Lauge-Hansen, it is the result of an exorotation force on the pronated foot.
- **Stage 1** - Avulsion of the medial malleolus or - ligamentous rupture
- **Stage 2** - Rupture of the anterior syndesmosis
- **Stage 3** - Fibula fracture above the level of the syndesmosis (this is the true Weber C fracture)
- **Stage 4** - Avulsion of the malleolus tertius or - rupture of the posterior syndesmosis.
- These fractures are identical to the fractures described by **Lauge-Hansen** as supination-adduction, supination-exorotation and pronation-exorotation

TREATMENT

- **Analgesia**
- **If displaced:**
 - o Reduction and immobilisation in a splint or cast.
 - o **Check neurovascular status pre-and post reduction**
 - o **Control X-ray post reduction**

Image



- Refer to ortho on call
- **If undisplaced:**
 - Below knee plaster of Paris.
 - Crutches
 - Advice on elevation
 - Referral to fracture clinic for follow up.

2. MAISONNEUVE FRACTURE

- **Maisonneuve fracture** is the combination of a spiral fracture of the **proximal fibula** and **unstable ankle injury** which could manifest radiographically by widening of the ankle joint due to distal **tibiofibular syndesmosis** and/or **deltoid ligament** disruption, or fracture of the medial malleolus.
- It is caused by pronation external-rotation mechanism. It requires **surgical fixation**.

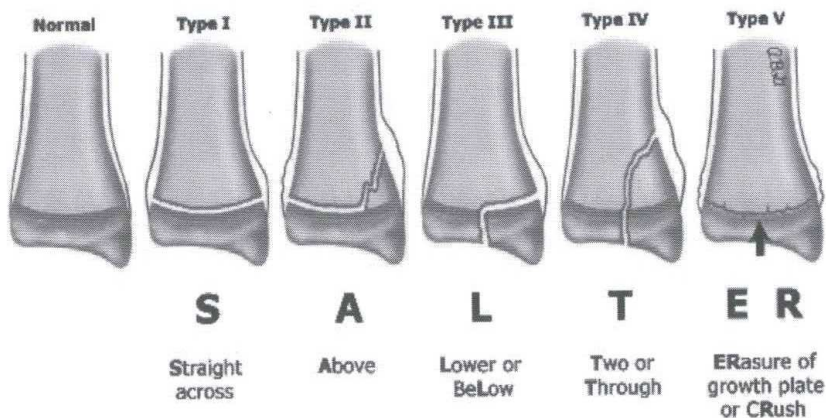


There is a spiral, comminuted fracture of the **proximal fibula** (A, black arrows).

There is also widening of the **distal tibio-fibular syndesmosis** (white arrows) and disruption of the ankle mortise with widening of the **tibio-talar joint** (white asterisk) and **talo-fibular joint** (black asterisk) which is compatible with a **Maisonneuve fracture**.

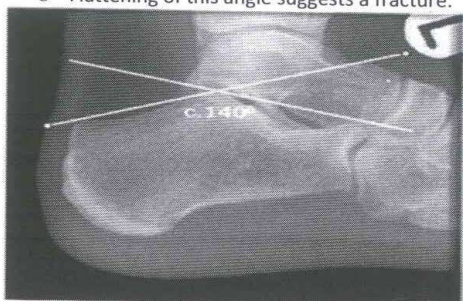
3. SALTER HARRIS FRACTURES

- The Salter Harris Classification of Paediatric fractures is as follow:



4. FRACTURED CALCANEUM

- o As discussed above, fractures may not be obvious on the **lateral X-ray** and it is important to obtain an **axial view**.
- o On the lateral X-ray, look at **Bohler's angle** which should be approximately 140° (or between $20-40^\circ$)
- o Flattening of this angle suggests a fracture.



Bohler's angle

Normal:
 $25^\circ-40^\circ$

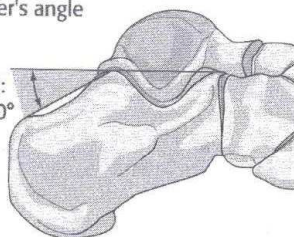


Figure 4.8.36. Bohler's angle

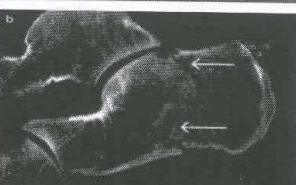
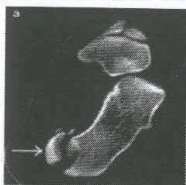
- Fractures of the calcaneum are the commonest tarsal fractures and usually classified into:
 - o **Extra-articular** – not involving the subtalar joint
 - o **Intra-articular fractures** – involving the subtalar joint

A. EXTRA-ARTICULAR CALCANEAL FRACTURES

- **Types:**
 - o They include fractures of:
 - The medial tubercle
 - The anterior process
 - The tuberosity
 - The sustentaculum tali
 - The body of the calcaneum posterior to the subtalar joint

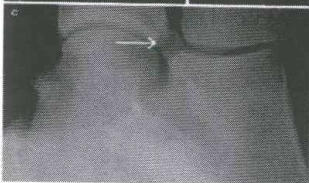
EXTRA ARTICULAR FRACTURES

Medial
Process #



Vertical
Tuberosity #

Anterior
Process #



Avulsion # at EDB
attachment



• **MANAGEMENT OF EXTRA-ARTICULAR CALCANEAL FRACTURES:**

- Treatment is usually conservative unless there is significant displacement, in which case open reduction and internal fixation (ORIF) will be undertaken.
- **Treatment in the ED includes:**
 - Analgesia
 - If there is any doubt about whether the fracture involves the subtalar joint, a **CT may be requested.**
 - **If displaced**, refer for orthopaedic opinion.
 - Otherwise:
 - **Support bandage** (eg wool and crepe) or **below-Knee plaster of Paris.**
 - **Crutches**
 - **Advice on elevation**
 - Referral to **fracture clinic** for follow up.
- Avulsion fractures of the Achilles tendon will need **ORIF.**
- **Prognosis:** These fractures are relatively minor and have a good prognosis

B. INTRA-ARTICULAR CALCANEAL FRACTURES

- Intra-articular calcaneal fractures are usually caused by a fall from a height onto the heel.
- In older patients with osteoporosis, the height may be as little as half a metre.
- Extra-articular fractures of the body and the medial tubercle are also caused by the same mechanism, though with lesser degrees of force. **Bilateral fractures are common** and, as discussed above, **calcaneal fractures may be associated with lumbar spine fractures.**
- There are several patterns of fracture but the exact patterns need not be known by emergency physicians as all these injuries will be referred to orthopaedic surgeons for further management.



Intra-articular fracture of the calcaneum. This image shows an intra-articular fracture of the calcaneum; the bone texture is abnormal and there are lucencies suggestive of a fracture but there are no obvious breaks in the cortex. Böhler's angle is grossly flattened.

• MANAGEMENT OF INTRAARTICULAR CALCANEAL FRACTURE:

- o Analgesia
- o Elevation foot
- o Patients will usually be admitted and investigated **further by CT**.
- o There are a variety of treatment options including reconstructive surgery.
- o **Prognosis:**
 - There is usually severe disruption of the subtalar joint and stiffness and arthritis of this joint requiring further surgery is very common.

LEARNING POINTS

- If you suspect a fractured calcaneum, ask for **specific calcaneal views**.
- When looking at a lateral ankle or foot X-ray, **always evaluate Bohler's angle**.

5. FRACTURED TALUS

- **Avulsion fractures of the talus** and **fractures of the talar dome** are classified as ankle injuries and are discussed in a different session.
- **Fractures of the body of the talus** are relatively uncommon injuries and are normally caused by major forces.
- The commonest cause for this is a road crash in which the car-driver's foot is forced backwards in a head-on collision. Injuries can also occur in a fall from a height.

Fractures of the neck of the talus are classified as:

- o **Type I:** undisplaced
- o **Type II:** displaced (however little) and associated with subluxation or dislocation of the subtalar joint
- o **Type III:** displaced with dislocation of the talus from the ankle joint
- Undisplaced fractures are easily missed and displaced fractures may be thought to be undisplaced and so **CT is valuable** in the assessment of talar injuries.
- As they are high velocity injuries, they may be associated with life-threatening injuries of the head and trunk and may be overlooked.
- Dislocation of the talus can occur with or without an associated fracture.



Figure 4.8.37. Undisplaced fracture of the waist of the talus (type I)



Figure 4.8.38. Displaced fracture of the talus (type II)

Management of Talar Fractures in the ED:

- **Truly undisplaced fractures:** can be treated in a below knee POP.
- All others need to be **referred to an orthopaedic surgeon** as an anatomical reduction is needed and this usually requires ORIF. If the skin is tight over the fracture, this is urgent.
- **Prognosis:**
 - o The major complication of these injuries is **avascular necrosis of the proximal part of the bone**.

6. FRACTURED NAVICULAR

- Minor avulsion fractures are common, often in association with a sprain of the ankle, and usually require no specific treatment.



Figure 4.8.39. Fractures navicular

- **Isolated fractures** of the navicular are uncommon. If they are undisplaced, they will normally be treated conservatively;
- **Displaced fractures** will need an orthopaedic opinion for consideration of ORIF.
- Fractures of the navicular may occur in association with dislocations of the mid-foot.
- Any significant injury in this area requires a lateral X-ray of the foot in addition to normal foot X-rays. If there is suspicion of a dislocation, **CT evaluation** is required.

7. FRACTURED CUBOID

- Minor avulsion fractures are common and usually require no specific treatment.
- Most fractures are **undisplaced** and will be treated conservatively.
- **Displaced fractures** may be part of a more complex foot injury and will need an orthopaedic review.

8. SUBTALAR DISLOCATION

- The subtalar joint is the joint between the talus and the calcaneum.
- If this joint dislocates, the forefoot stays attached to the calcaneum and so the talonavicular joint also dislocates.
- Subtalar dislocation occurs in excessive inversion or eversion and can occur medially or laterally. It may be associated with a fracture of the lateral malleolus.

MANAGEMENT

- Ideally the ankle and foot should be X-rayed to confirm the diagnosis.
- However, if there is neurovascular impairment or if the skin is stretched and there is concern that tightness of the skin may risk skin necrosis, it is common practice to try to reduce significantly displaced ankle and foot injuries before X-ray.
- If a displaced fracture is reduced before X-ray, the fracture is still visible and so a diagnosis is still possible but if a dislocation is reduced before X-ray, the subsequent X-ray may be normal and it may be difficult to establish the true diagnosis.



Lateral subtalar dislocation (and fracture of the lateral malleolus)

Medial subtalar dislocation

- These injuries should be **reduced under sedation or general anaesthesia** and **immobilised in a below knee POP**. They should be followed up by an orthopaedic surgeon.

9. MIDTARSAL DISLOCATION

- In a midtarsal dislocation, the cuboid and navicular dislocate from the talus and calcaneum. The joint may dislocate medially (with an adduction force) or laterally (with an abduction force). These dislocations may be associated with fractures of the tarsal bones (particularly the navicular) or with smaller avulsion fractures.
- In major foot injuries always obtain a lateral X-ray of the foot in addition to the usual AP and oblique views. Similarly, if standard X-rays show a fracture of the navicular, obtain a lateral X-ray of the foot.
- If there is a significant mechanism of injury and the patient clinically has a fracture with much swelling but the X-rays appear normal or relatively normal, consider doing a CT.

10. LISFRANC INJURY or TARSO-METATARSAL DISLOCATION

- The tarso-metatarsal joint is also known as the **Lisfranc joint** and so dislocations at this site are also known as **Lisfranc injuries**.
- These are commonly missed but the clue to their diagnosis is **abnormalities in the alignment of the metatarsals with the tarsal bones**
- **The discoloration on the bottom of the foot is very suggestive of a Lisfranc injury.**

MANAGEMENT:

- These injuries need orthopaedic referral.
- Most will be investigated with a **CT** and require internal fixation.
- Not all injuries at this joint are obvious on initial X-rays. If it is suspected clinically but X-rays are normal, the patient should be kept under review and consideration given for a CT.

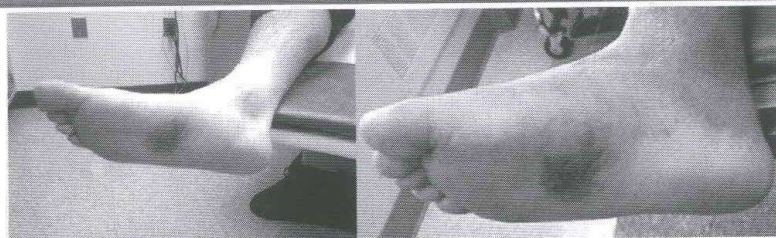


Figure 4.8.40. Plantar Ecchymosis



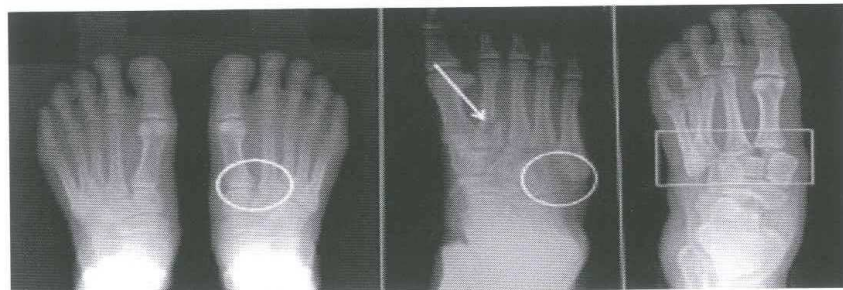
Figure 4.8.41. Normal alignment between tarsus and metatarsals



Figure 4.8.42. Severe tarso-metatarsal dislocation



Figure 4.8.43. Subtle Lisfranc injury



Left: This is a subtle injury to the midfoot with widening between the first and second metatarsals (circle), compared with the normal foot on the left.

Center: This x-ray shows a fracture of the second metatarsal (arrow) and a fracture of the cuboid (circle).

Right: This shows a very severe injury of the foot from a high-energy event. It has resulted in a complete dislocation of the entire midfoot (box). Because no bones have been broken, a fusion may be recommended, given the high risk for future arthritis.

11. FRACTURES OF THE FIFTH METATARSAL

- Fractures of the 5th metatarsal base in association with an inversion injury of the ankle are avulsion fractures occurring at the insertion of the tendon of **peroneus brevis**.
- They are normally treated symptomatically with either a supportive bandage or plaster, with or without crutches (depending on the patient's mobility).
- Most fractures heal quickly but occasionally go to non-union.
- This only needs treatment if it is symptomatic.
- These fractures must also be differentiated from fractures at the base of the shaft.
- These are usually **stress fractures** and are commonly called **Jones fractures** though they can occur as a result of direct trauma.
- These are important as there is a significant incidence of non-union and so they are normally treated in plaster and should be referred for orthopaedic follow-up. The apophysis at the base of the 5th metatarsal in children may be mistaken for a fracture.
- However, the apophyseal line is longitudinal (**parallel to the metatarsal**) whereas fractures are transverse (**perpendicular to the metatarsal**).
- The apophysis may be fragmented i.e. an apophysis and a fracture can co-exist.
- Fractures of the shaft of the metatarsal are treated symptomatically.



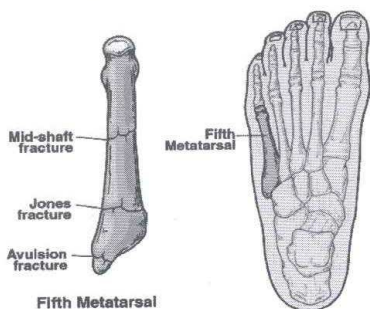
Fig 4.8.44. A normal apophysis at the base of the 5th metatarsal in a child.



Oblique radiograph of the foot shows a transverse avulsion fracture (arrow) of the 5th metatarsal bone with displacement and extension into the cuboid-metatarsal joint. Note a nearby 'os peroneum' (arrowhead).

Fractures, foot. Proximal fifth metatarsal avulsion fracture (also termed **pseudo-Jones, tennis, or dancer fracture**).

12. JONES FRACTURE



- Transverse fracture of shaft of little metatarsal.
- Very different to pull off fracture as above.
- **Unstable** as peroneus (brevis) tendons distract fracture and mal/non-union likely.
- Treat in POP and refer fracture clinic.

13. STRESS FRACTURES



- Sports related insidious onset midfoot pain; Point tender over dorsum navicular ("N spot")
- Tender or medial plantar arch (less specific); Pain with passive eversion and active inversion. Difficult to see on plain views (? bone scan or CT)
- **Management:**
 - Strict non-weight bearing POP and Fracture Clinic Referral
 - Analgesia, physio rehab back to sports

14. FRACTURES OF OTHER METATARSALS

- Fractures of a single metatarsal (2nd, 3rd or 4th) are usually well splinted by the other, intact, metatarsals and require symptomatic treatment only.
- However, fractures of the 1st metatarsal may displace and **need internal fixation**.
- If there are multiple metatarsal fractures, this allows each fracture to displace.
- These patients need orthopaedic admission both for elevation and treatment of the associated soft tissue swelling and for consideration of internal fixation of the fractures.
- Fractures at the bases of the metatarsals (except for 5th) may be associated with injuries of the tarso-metatarsal joint.

15. TOES INJURIES

- Most toe fractures will be caused either by dropping a weight on the foot or by stubbing the toe.
- **An undisplaced fracture** requires no specific treatment but will usually be treated with neighbour strapping for a few weeks and advice on analgesia and footwear.
- Most patients seem more comfortable in sandals but some prefer wearing walking boots or similar as they are less likely to knock their toe.
- **Displaced fractures** may require manipulation followed by neighbour strapping.
- Displaced fractures of the big toe are more serious than injuries of the other toes. These **may need internal fixation**.
- It has been argued that X-rays of clinically undisplaced injuries of the toe are unnecessary as they do not alter treatment. This is only true as long as the toe is examined carefully as it is important not to miss a dislocation of the toe as these need reduction.

16. SUBUNGUAL HAEMATOMA

- A subungual haematoma is usually caused by a weight falling onto the toe which may also cause a fracture.
- The pressure from it often causes significant pain and this can be significantly relieved by trephining the nail to allow the release of blood.
- However, there is no evidence that this treatment is better than no treatment.
- It is sometimes argued that patients with an underlying fracture should be given antibiotics as the act of trephining converts a closed fracture to an open one.
- *There is no evidence to support this approach.*

17. DISLOCATION OF THE TOES

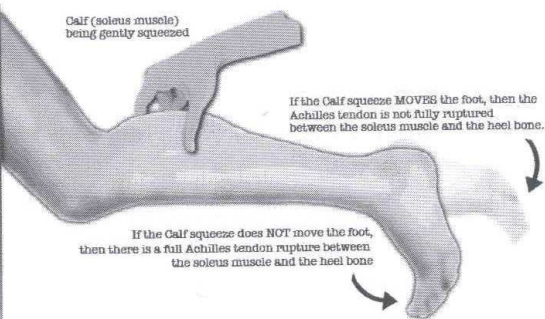
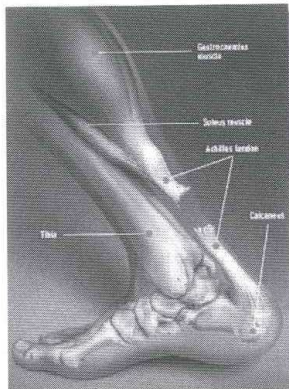
- Dislocations usually occur at the metatarso-phalangeal joint or the inter-phalangeal joint of the big toe.
- They should be reduced under local anaesthesia and a post reduction X-ray obtained.

18. ACHILLES TENDON RUPTURE

- **RISK FACTORS**
 - *Steroid or Quinolone use*
 - *Rheumatoid arthritis/ SLE/ Gout*
 - *Renal failure*
 - *Hyperparathyroidism*
 - *Hyperlipoproteinaemia*
- **Clinical**
 - Observe fracture foot may not rest in natural plantar-flexion
 - **Palpable step** in Achilles Tendon
 - **Thompson test** - lie prone and calf squeeze produces plantar flexion in normal individuals.
 - **Matles test** - lie prone, knees flexed 90°, gravity makes fracture side ankle more dorsiflexed.

• Differential

- Server's (calcaneal apophysitis) in teenagers
- Peroneal tendonopathy or dislocation
- Retrocalcaneal bursa, Os trigonum syndrome
- Ankle OA, Systemic arthritis (check other side)
- Sural neuroma (or referred pain from sacral roots)



Calf squeeze test for Achilles tendon rupture

• Management

- Refer to on call orthopaedic team
- Operative repair is preferable to conservative management
- If conservative Mx consider prophylactic anticoagulation (LMWH) particularly if high risk of VTE or prior DVT.

PAEDIATRIC PRESENTATIONS

11 QUESTIONS

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CHAPTER 1. APNOEA, STRIDOR AND AIRWAY OBSTRUCTION

I. BRONCHIOLITIS

• Introduction

- Bronchiolitis is the most common disease of the lower respiratory tract during the first year of life. Bronchiolitis is typically caused by a virus.
- **Respiratory syncytial virus (RSV)** is the most common cause.
- It usually presents with **cough** with **increased work of breathing**, and it often **affects a child's ability to feed**.
- *In primary care, the condition may often be confused with a common cold, though the presence of lower respiratory tract signs (wheeze and/or crackles on auscultation) in an infant in mid-winter would be consistent with this clinical diagnosis.*
- The symptoms are usually mild and may **only last for a few days**, but in some cases the disease can cause severe illness.
- There are several individual and environmental risk factors that can put children with bronchiolitis at increased risk of severe illness.

Healthcare professionals should be aware of the increased need for hospital admission in infants with the following:

- Pre-existing lung disease, congenital heart disease, neuromuscular weakness, immune-incompetence
- Age < 6 weeks (corrected)
- Prematurity
- Family anxiety
- Re-attendance
- Duration of illness is less than 3 days and Amber- may need to admit

Signs and Symptoms can include:

- Rhinorrhoea (Runny nose)
- Cough
- Poor feeding
- Vomiting
- Pyrexia
- Respiratory distress
- Apnoea
- Inspiratory crackles ± wheeze
- Cyanosis

• Diagnostic Criteria

- Diagnose bronchiolitis if the child has:
 - **A coryzal prodrome** lasting 1 to 3 days, followed by:
 - Persistent **cough** and either
 - **Tachypnoea** or **chest recession** (or both) and either
 - **Wheeze** or **crackles** on chest auscultation (or both).
- When diagnosing bronchiolitis, take into account that the following symptoms are common in children with this disease:
 - Fever (in around 30% of cases, usually of less than 39°C)
 - Poor feeding (typically after 3 to 5 days of illness).
- When diagnosing bronchiolitis, take into account that young infants with this disease (in particular those under 6 weeks of age) may present **with apnoea without other clinical signs**.
- Consider a diagnosis of **Pneumonia** if the child has:
 - Bronchiolitis in children: high fever (over 39°C) and/or
 - Persistently focal crackles.

- o Think about a diagnosis of **viral-induced wheeze** or **early-onset asthma** rather than bronchiolitis in older infants and young children if they have:
 - Persistent wheeze without crackles or
 - Recurrent episodic wheeze or
 - A personal or family history of atopy.
- o Take into account that the above conditions are unusual in children under 1 year of age

• **ADMISSION CRITERIA**

- o Admit to hospital if **life threatening symptoms (RED FLAGS)**:
 - *Unable to rouse*
 - *Apnoea (observed or reported)*
 - *Persistent SPO₂ <92% when breathing air*
 - *Inadequate oral fluid intake: < 50% fluid intake over 2-3 feeds*
 - *Significant reduced urine output*
 - *Persisting severe respiratory distress: grunting, marked chest recession, or a respiratory rate of over 70 breaths/minute, cyanosis*
 - *Pale, mottled skin with CRT > 3sec*
 - *Presence of risk factors*

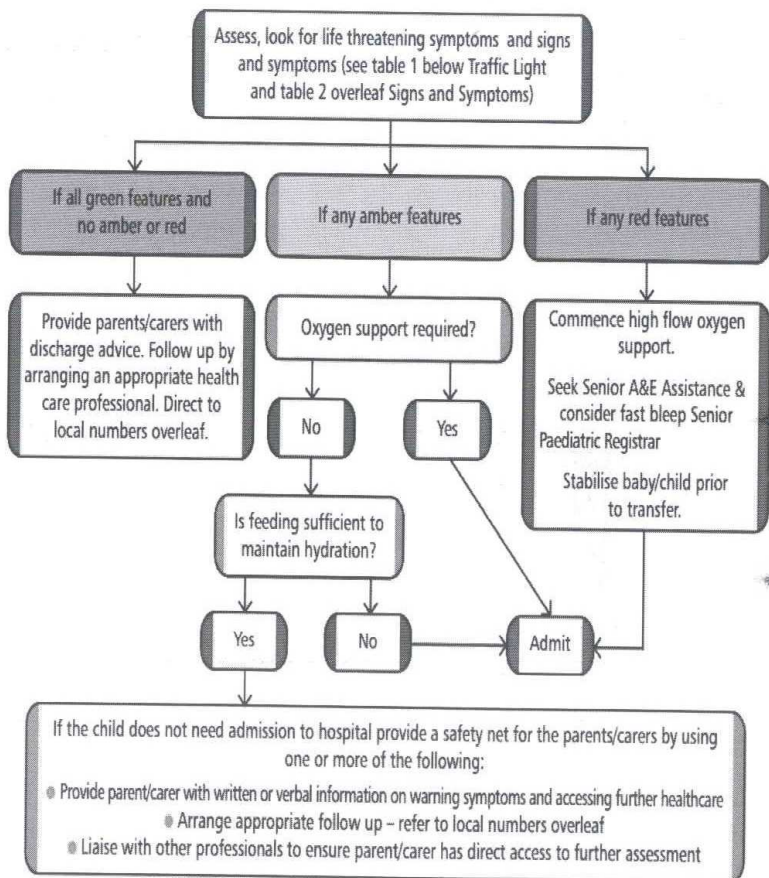
Traffic light system for identifying severity of illness

	GREEN-LOW RISK	AMBER-INTERMEDIATE RISK	RED-HIGH RISK
Behaviour	<ul style="list-style-type: none"> • Alert • Normal 	<ul style="list-style-type: none"> • Irritable • Not responding normally to social cues • Decreased activity • No smile 	<ul style="list-style-type: none"> • Unable to rouse • Wakes only with prolonged stimulation • No response to social cues • Weak, high pitched or continuous cry • Appears ill to a healthcare professional
Skin	<ul style="list-style-type: none"> • CRT ≤2secs • Normal colour skin, lips & tongue 	<ul style="list-style-type: none"> • CRT 2-3secs • Pale/mottled • Pallor colour reported by parent/ carer • Cool Peripheries 	<ul style="list-style-type: none"> • CRT over 3 secs • Pale/mottled/ ashen blue • Cyanotic lips and tongue
Resp rate	< 12mths: <50bpm > 12 mths: < 40bpm No respiratory distress	< 12mths: 50-60bpm > 12 mths: 40-60bpm	All ages > 60bpm
SATS in air	95% or above	92-94%	<92%
Chest recession	None	Moderate	Severe
Nasal flaring	Absent	May be present	Present
Grunting	Absent	Absent	Present
Feeding/ Hydration	<ul style="list-style-type: none"> • Normal • No vomiting 	<ul style="list-style-type: none"> • 50-75% fluid intake over 3-4 feeds ± vomiting. • Reduced urine output 	<ul style="list-style-type: none"> • < 50 % fluid intake over 2-3 feeds ± vomiting. • Significantly reduced urine output
Apnoea	Absent	Absent	Present

ED MANAGEMENT

- o The management of bronchiolitis depends on the severity of the illness.
- o In most children bronchiolitis can be managed at home by parents or carers.
- o *Do not use any of the following to treat bronchiolitis in children: antibiotics, hypertonic saline, adrenaline (nebulised), salbutamol, montelukast, ipratropium bromide, systemic or inhaled corticosteroids*

Management within Hospital Setting



II. CROUP (LARYNGOTRACHEOBRONCHITIS)

INTRODUCTION

- The word **stridor** is derived from the Latin "stridulus", which means creaking, whistling or grating. It is a sign of airway obstruction.
- Croup is a syndrome consisting of a "**barking**" cough, **stridor**, **hoarseness** and **varying degrees of difficulty breathing**.
- The volume of stridor does NOT correlate with the degree of obstruction.
- The underlying pathology is inflammation of the pharynx, larynx, trachea or bronchi.
- It is subglottic inflammation and swelling that compromises the airway in croup.
- Poiseuille's Law states that if the radius of the airway is halved then the resistance in the airflow increases by 16-fold.
- A small reduction in the diameter of the airway dramatically reduces the airflow and the child can rapidly deteriorate.

ETIOLOGIES

- Croup is the most common cause of acute stridor.
- In 80% of cases the cause of croup is viral and the majority are **Parainfluenza viruses**.
- Other viruses that cause croup are: *Adenovirus, RSV, Measles, Coxsackie, Rhinovirus, Echovirus, Reovirus and Influenza A and B.*

CLINICAL PRESENTATION OF CROUP

THE WESTLEY CROUP SCORE

Score	Stridor	Retractions	Air Entry	SaO ₂ <92%	Level of consciousness
0	None	None	Normal	None	normal
1	Upon agitation	Mild	Mild decrease		
2	At rest	Moderate	Marked decrease		
3		Severe			
4				Upon agitation	
5				At rest	Decreased

- Children with croup can be divided into four levels of severity:
 - **Mild:** croup score 0-2
 - **Moderate:** croup score 3-5
 - **Severe:** croup score 6-11
 - **Impending respiratory failure:** croup score 12-17
- 85% of children have mild croup.
- 5% of children are admitted into hospital and of these 1-3% require intubation.
- Uncommon complications include **pneumonia** and **bacterial tracheitis**.

DIFFERENTIAL DIAGNOSIS OF ACUTE STRIDOR

- Excluding croup, the most likely causes of acute stridor are:
 - *Foreign body aspiration,*
 - *Angioedema,*
 - *Abscess (peri-tonsillar or parapharyngeal),*
 - *Epiglottitis*
 - *Bacterial tracheitis.*

CAUSES OF AIRWAY OBSTRUCTION	EXAMPLES
Obstructive	Foreign body Post-tonsillectomy bleed Burns Facial or laryngeal trauma (e.g. compressive haematomas) Tongue (in an obtunded patient)
Infective	Epiglottitis Croup Bacterial tracheitis Diphtherias Whooping cough Retropharyngeal abscess
Allergic	Anaphylaxis (angioedema)

INVESTIGATION STRATEGIES

- Croup is essentially a **clinical diagnosis** and no investigations are required to make a diagnosis; ABG analysis and chest x-ray may be helpful in assessing severity and potential complications.

MANAGEMENT OF CROUP IN THE ED

- Make the child **comfortable** (avoid agitating the child).
- Oxygen** if saturation is less than 92% on air.
- Dexamethasone 0.15 mg/kg po**
- Prednisolone (1 mg/kg).**
- If the child is vomiting, **Nebulised budesonide 2 mg** can be used.
- Nebulised Adrenaline** is **only used** in children with severe and life-threatening croup: **Adrenaline 1: 1,000 0.5ml/kg Nebs** maximum 5 mL.
- Referral severe cases and complications to a senior paediatric trained doctor and early consideration of PICU involvement is essential.
- Antibiotics not indicated (mostly viral aetiology).
- The main stay of treatment for croup is corticosteroids, which take between 2 and 4 hours to have a clinical effect.*

Monitoring

- The respiratory rate, work of breathing, oxygen saturation and pulse rate should be carefully monitored.
- The work of breathing, respiratory rate, volume of stridor and pulse rate should decrease if the treatment is working.

Disposal

- Mild croup:** can be discharged home **following a single dose of dexamethasone**
- Moderate croup:** observe for a **minimum of four hours** following a dose of dexamethasone and then re-assessed.
- Severe croup:** must be admitted into hospital.
- In children discharged home advice must be given to a parent and documented in the notes.

OTHER CAUSES OF ACUTE STRIDOR & MANAGEMENT**1. FOREIGN BODY**o **Presentation:**

- The usual history obtained is of sudden onset coughing, retching and choking. See the management of choked child below.
- Partial obstruction above or at the vocal cords causes inspiratory stridor, a change in voice, cough and dyspnoea.
- Partial obstruction of the lower airway in addition to cough and dyspnoea may cause a pneumothorax, pneumomediastinum or surgical emphysema.
- Findings on examination will depend on the site of the obstruction and may include: cough, wheeze, stridor and signs pneumonia.

o **Investigations:**

- An inspiratory **chest x-ray** may be normal, whilst an expiratory film may demonstrate air trapping.

o **Treatment:**

- It is by removal of the foreign body by **bronchoscopy** under general anaesthetic.

2. ANGIOEDEMA

- o Angioedema, with or without urticaria, is classified as allergic, hereditary, or idiopathic. (see Anaphylaxis notes)
- o Airway compromise is caused by vasodilatation and associated oedema.
- o Treatment of allergic and idiopathic angioedema is with IM adrenaline, oxygen, steroids, H1 and H2 blockers, IV fluids and consideration of intubation.
- o If adrenaline is required, then all children must be admitted for observation due to the risk of re-occurrence after six hours.
- o On discharge children, should be referred to an allergy specialist, receive training on the use of an adrenaline auto-injector (e.g. EpiPen or Anapen) and be discharged with two adrenaline auto-injectors, one of which should be kept at school.
- **In Hereditary angioedema (HAE)** (autosomal dominant disorder of C1 esterase inhibitor):
 - o Oedema formation is related to the reduction or dysfunction of C1 inhibitor which results in the release of bradykinin and C2-kinin mediators.
 - o This enhances vascular permeability and leads to extra-vascular fluid shifts.
 - o Approximately 40% of people with HAE present with the first episode before the age of 5 years and 75% present before age 15 years.
 - o **Treatment of HAE:**
 - **C1 inhibitor concentrate** is the treatment of choice.
 - Clinical improvement is seen within 15 to 60 minutes.
 - A repeat dose may be required if symptoms are not relieved within an hour or progress.
 - If C1 inhibitor concentrate is not available, then **fresh frozen plasma** or solvent detergent treated plasma (**Octaplas**) can be used.
 - **HAE does not respond to adrenaline.**

Dose of C1 inhibitor concentrate	
Weight	Dose
<50Kg	10U/Kg
50-100Kg	1000U
>100Kg	1500U

3. ABSCESS

- o In **retropharyngeal abscess**, the bacteria most commonly identified are *streptococcus pyogenes*, *staphylococcus aureus*, *haemophilus influenzae* and *neisseria species* and *anaerobes*.
- o Presentation is with fever, sore throat and poor oral intake. Examination may reveal a neck mass, fever, cervical adenopathy, neck stiffness or torticollis, agitation, lethargy, drooling, trismus and stridor.
- o In stable patients **lateral soft tissue x-rays** can show an enlarged prevertebral soft tissue shadowing. Children with airway compromise must be admitted for close monitoring with **urgent I&D** of the abscess.

4. EPIGLOTTITIS

- o Following the introduction of the Haemophilus influenza type b (Hib) vaccination in 1992, childhood epiglottitis has become rare.
- o It can also be caused by the same aerobes that cause peri-tonsillar abscesses.
- o Children who are fully immunised can still get Hib culture positive epiglottitis.
- o There is a rapid onset of pyrexia, sore throat, muffled speech, drooling and stridor.
- o The child usually looks unwell, sitting forwards, mouth open, drooling and with their tongue protruding.
- o Management of this condition remains controversial:
 - The cornerstone is **not to distress** the child as this can precipitate complete airway obstruction.
 - **Oxygen** should be administered if the child is hypoxic.
 - In the first instance, intravenous antibiotics should be administered, if IV access can be achieved without distressing a younger child.
 - **A third-generation cephalosporin** is a reasonable choice.
 - Children under six years of age require **urgent intubation**, ideally in theatre by an experienced anaesthetist with an ENT surgeon present.
 - If there is no time to transfer the child to theatre, then a difficult intubation trolley and cricothyroidotomy kit must be accessible.
 - In those over the age of six years observation may be an option following consultation with an ENT and PICU consultant.
- o The average time for children to remain intubated is **48 to 96 hours**.
- o Extubation occurs when direct visualisation of the epiglottis confirms that the inflammation of the epiglottis and surrounding tissues has resolved.

5. TRACHEITIS

- o **Bacterial tracheitis** may occur at any age. In the early phase patients may present similarly to croup however there is a failure to respond or a transient response to steroids and/or nebulised adrenaline and the condition worsens.
- o In this condition the larynx, trachea and bronchi can become obstructed with purulent debris.
- o There is an adherent pseudomembrane that forms over the tracheal mucosa that can slough off causing an obstruction.
- o There is normally a preceding upper respiratory tract infection for a couple of days, followed by a rapid deterioration with a pyrexia and respiratory distress.
- o There is a cough producing copious secretions and retrosternal pain.
- o *There is no dysphagia or drooling unlike epiglottitis.*

- The most common causative organisms are *Staphylococcus aureus* (41%), *Haemophilus influenzae* (18%), *Streptococcus pneumoniae* (15%), *Moraxella catarrhalis* (13%) and *Streptococcus pyogenes* (9%).
- **Treatment**
 - IV antibiotics.
 - Endotracheal intubation is often needed for airway control, management of respiratory failure and pulmonary toilet.
 - Young children can deteriorate quickly due to the smaller size of the airway.
- Full recovery with no long-term morbidity is expected in the vast majority of children.
- The mean length of stay in hospital varies with reports between 3 and 12 days.
- **Complications of tracheitis**
 - **Most frequent:** Pneumonia
 - **Less common complications:**
 - Acute Respiratory Distress Syndrome (ARDS)
 - Septic shock
 - Pulmonary oedema / Pneumothorax
 - Cardiorespiratory arrest
 - Long-term morbidity associated with bacterial tracheitis is minimal.
 - As treatment in the acute phase of the illness frequently requires insertion of an **endotracheal tube** into an inflamed airway, the potential for the subsequent development of subglottic stenosis is well recognised.

Comparison between Croup, Epiglottitis and Tracheitis.

	Croup	Epiglottitis	Tracheitis
Incidence	Common	Rare	Rare
Age	6 months - 3 years	2 - 7 years	6 months - 14 years
Aetiology	Viral	Bacterial	Bacterial
Speed of onset	Slow	Very rapid	Rapid
Fever	Rarely >39 degrees	Normally >39 degrees	Normally >39 degrees
Cough	Barking	Suppressed	Present
Voice	Hoarse	Muffled	Hoarse
Position	Supine	Sitting forward, neck extended	Supine
Neck x-ray AP	Steeple sign*	Normal	Steeple sign*
Neck x-ray Lateral	Normal	Thumb print	Hazy
Response to adrenaline	Very good	No response	Partial or no response

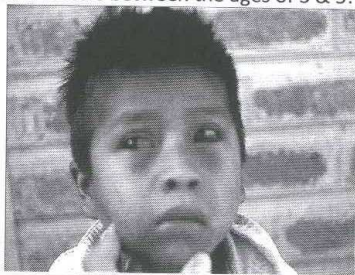
***Steeple sign:** On anteroposterior radiographs of the soft tissue of the neck the lateral convexities of the subglottic trachea are lost and narrowing of the subglottic lumen produces an inverted "V" pattern, resembling a church steeple.

6. LARYNGOMALACIA

- It is the commonest congenital laryngeal abnormality and a relatively common cause of stridor in infancy. An abnormality of the laryngeal cartilages causes the larynx to be soft and floppy and collapse during inhalation causing partial airway obstruction.
- Presentation tends to occur within a few weeks of birth with an inspiratory stridor that is worse when feeding, agitated or lying in the supine position.
- The stridor is often described as being '**high-pitched**' or '**crowling**'. The stridor can be worsened by a co-existing coryza and tends to initially worsen before spontaneously resolving within the **first 18-14 months of life**.
- The diagnosis can be confirmed by **flexible laryngoscopy** and treatment is rarely required.

III. WHOOPING COUGH

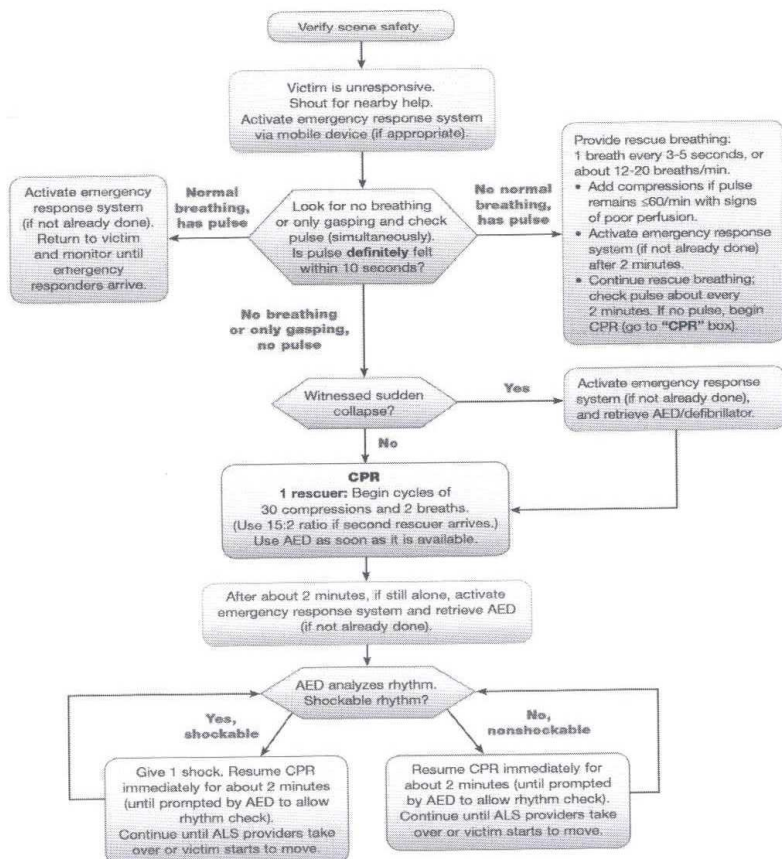
- Pertussis is also known as **whooping cough** because of its characteristic coughing sound.
- Whooping cough (pertussis infection) is caused by **Bordetella pertussis** and is a notifiable disease.
- This disease is preventable by childhood immunisation.
- It is a highly infectious bacterial disease of the respiratory tract and spread by droplet transmission.
- The incubation period is **7–10 days** but the infectious period can be from 4 days before to 3 weeks after the onset of typical paroxysms.
- The highest incidence is in infants who are not immunized or too young to be fully protected.
- School children are often the source of infection for younger siblings at home.
- Infection can also occur in adolescents and adults, even if previously immunized, because immunity wanes over time.
- **Vaccination**
 - In the UK acellular pertussis vaccine is given in the primary course with diphtheria, tetanus, polio and Hib (as DtaP/IPV/Hib), given at aged 2, 3, & 4 months of age.
 - A further booster dose is given with the preschool boosters between the ages of 3 & 5.
- **Clinical presentation**
 - Initial symptoms include coryza and cough.
 - Gradually the cough progresses to severe coughing bouts which can be prolonged.
 - Not all children have the characteristic 'whoop' (inspiratory noise) at the end of a coughing bout, and some cough spasms may be followed by periods of vomiting.
 - These coughing episodes can be severe and may result in **subconjunctival and periorbital haemorrhages**.
- **Complications**
 - Pneumonia, seizure, encephalopathy, weight loss and death.
 - Complications are most likely to occur in young infants among who the most common cause of pertussis related deaths is **secondary bacterial pneumonia**.
 - Pertussis can occur in previously immunized and infected individuals, but immunization and prior infection attenuate the clinical picture.
- **Investigation**
 - Confirmation of the diagnosis is via **PCR** and **serological testing** because the viral culture lacks sensitivity.
 - Other investigations should be directed as for a suspected pneumonia.
- **Management of Pertussis in the ED**
 - Antibiotic prophylaxis may be of value for unvaccinated household contacts of cases, particularly in infants <6 months of age, **if given within 21 days of onset of the first case**.
 - **Macrolide antibiotics** (azithromycin, clarithromycin, and erythromycin) are recommended for the treatment of pertussis in people aged ≥1 month.
 - For infants aged <1 month, **Azithromycin** is the preferred antibiotic.



Subconjunctival and periorbital haemorrhages

CHAPTER 2. CARDIO-RESPIRATORY ARREST**I. PAEDIATRIC CARDIAC LIFE SUPPORT**

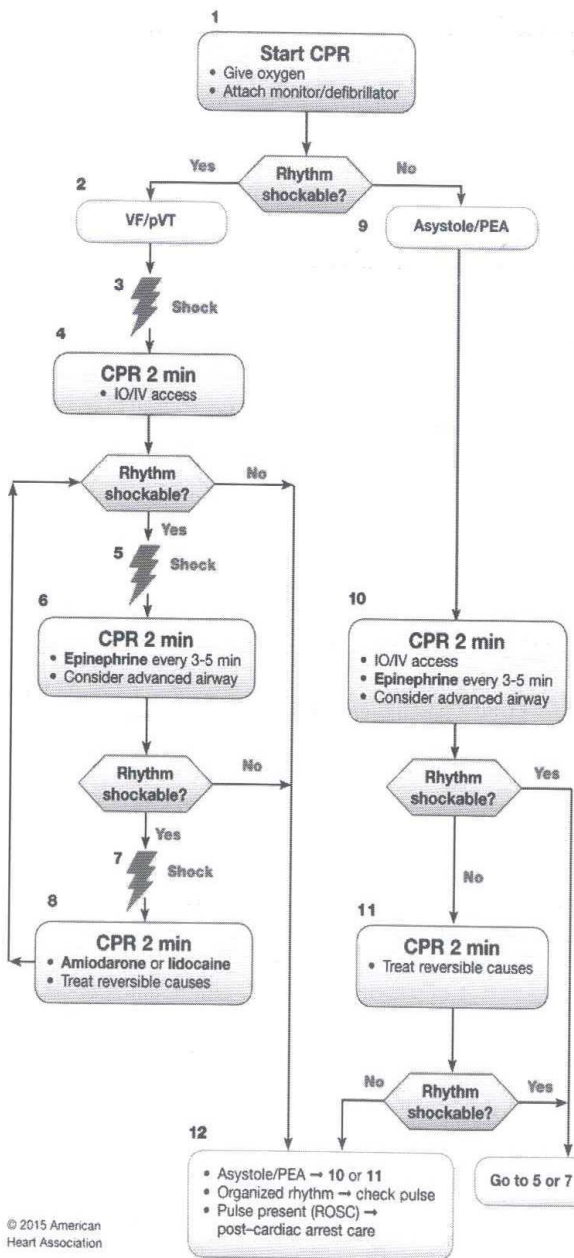
- o A **newborn** is a child just after birth.
- o A **neonate** is a child in the first 28 days of life.
- o An **infant** is a child under 1 year.
- o A **child** is between 1 year and puberty.

1. PAEDIATRIC BLS AND ACLS**BLS Healthcare Provider****Pediatric Cardiac Arrest Algorithm for the Single Rescuer—2015 Update**

© 2015 American Heart Association

Figure 5.2.1. Paediatric advanced life support algorithm

Pediatric Cardiac Arrest Algorithm—2015 Update



CPR Quality

- Push hard ($\geq \frac{1}{2}$ of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 15:2 compression-ventilation ratio.

Shock Energy for Defibrillation

First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥ 4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy

- **Epinephrine IO/IV dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes.
If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.
- **Lidocaine IO/IV dose:** Initial: 1 mg/kg loading dose. Maintenance: 20-50 mcg/kg per minute infusion (repeat bolus dose if infusion initiated >15 minutes after initial bolus therapy).

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

2. DRUGS USED IN CPR• **Adrenaline**

- Adrenaline 1:1000=1mg/ml
- Adrenaline 1:10 000= 0.1mg/ml or 100mcg/ml or 1mg/10ml
- **Children CPR dosage: IV/IO Adrenaline 10 micrograms/kg.**
- Subsequent doses of adrenaline are given **every 3–5 min.**
- Do not use higher doses of IV adrenaline in children, it may worsen outcome.

• **Amiodarone**

- Amiodarone 150mg/3ml= **50mg/ml** (450mg/9ml and 900mg/18ml)
- **Children CPR dosage:** In the treatment of shockable rhythms:
 - Initial IV bolus dose of **Amiodarone 5 mg/kg after the third defibrillation.**
 - Repeat the dose after the **fifth shock if still in VF/pVT.**
 - If defibrillation was successful but VF/pVT recurs, amiodarone can be repeated (unless two doses have already been given) and a continuous infusion started.
- Amiodarone can cause **thrombophlebitis** when injected into a peripheral vein and, ideally, should be delivered via a central vein.
- If central venous access is unavailable (likely at the time of cardiac arrest) and so it has to be given peripherally, flush it liberally with **0.9% sodium chloride or 5% glucose.**

• **Atropine**

- Atropine is effective in increasing heart rate when bradycardia is caused by excessive vagal tone (e.g. after insertion of nasogastric tube).
- The dose is **20 mcg/kg.**
- There is no evidence that atropine has any benefit in asphyxial bradycardia or asystole and its routine use has been removed from the ALS algorithms.

Magnesium

- **Indications during CPR (only if):**
 - Hypomagnesaemia
 - Polymorphic VT (torsade de pointes).

• **Calcium**

- **Indications during CPR (only if):**
 - Hyperkalaemia,
 - Hypocalcaemia
 - Overdose of calcium-channel-blocking drugs
- High plasma concentrations achieved after intravenous injection may be harmful to the ischaemic myocardium and may also impair cerebral recovery.

• **Sodium bicarbonate**

- **8.4% NaHCO₃- 1 mEq/mL Injection=> 50 mL Single Dose Vial (50mEq/50ml);**
- **1 mEq = 84mg NaHCO₃-**
- Cardiorespiratory arrest results in combined respiratory and metabolic acidosis, caused by cessation of pulmonary gas exchange and the development of anaerobic cellular metabolism respectively. The best treatment for acidaemia in cardiac arrest is a combination of effective chest compression and ventilation (high quality CPR).
- The routine use of sodium bicarbonate in CPR is not recommended.
- **Indications during CPR (only if):**
 - Hyperkalaemia
 - Arrhythmias associated with TCA overdose

○ RISK OF ADMINISTRATION OF SODIUM BICARBONATE:

- It generates carbon dioxide, which diffuses rapidly into the cells, exacerbating intracellular acidosis if it is not rapidly cleared via the lungs.
- It produces a negative inotropic effect on an ischaemic myocardium.
- It presents a large, osmotically active, sodium load to an already compromised circulation and brain.
- It produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues.

● Fluids in CPR

- If hypovolaemia is suspected, give **IV or IO fluids rapidly (20 mL/kg boluses)**.
- In the initial stages of resuscitation there are no clear advantages in using colloid solutions, whatever the aetiology, so use isotonic saline solutions for initial volume resuscitation. **Do not use dextrose-based solutions for volume replacement**; these will be redistributed rapidly away from the intravascular space and will cause hyponatraemia and hyperglycaemia, which may worsen neurological outcome.

3. POST-RESUSCITATION CARE

● Immediate Post cardiac arrest treatment:

○ Use of ABCDE approach:

- **A & B:** Controlled oxygenation (Sat 94-98%), Advanced Airway, use waveform capnography and ventilate lungs to Normocapnia
- **C:** ECG, IV access & Investigations, CXR, IV fluids if hypotension and Inotropes
- **D:** Blood Glucose Control, Treat precipitating causes
- **E:** Temperature control (32-36°C)

4. SUPRAVENTRICULAR TACHYCARDIA IN INFANTS

- ED management is dependent on the patient stability at presentation.

● IN A STABLE PATIENT

- **Vagal maneuvers** at this age include icing the face, avoiding the nares.
- If unsuccessful, IV access should be established, and adenosine **100mcg/kg IV push** followed immediately by flush should be administered (maximum of 6 mg/kg).
- If SVT persists then a second dose of adenosine **200mcg/kg IV** (maximum of 12 mg/kg).
- After a further two minutes another dose of **300 mcg/kg adenosine** should be given
- If the child remains in stable SVT despite these measures then the guidelines recommend that following be considered:
 - **Adenosine 400-500 mcg/kg**
 - **Synchronous DC shock**
 - **Amiodarone**

● AN UNSTABLE PATIENT

- Without IV access should be treated with **synchronized cardioversion (1 - 2 J/kg)**.
- If there is established IV access and adenosine is readily available, then the initial cardioversion may be attempted pharmacologically.
- If the SVT is unresponsive to adenosine or synchronized cardioversion or if a wide QRS is suspected, then **amiodarone 5 mg/kg IV over 20-60 minutes** may be administered.
- Alternatively, **Procainamide 15 mg/kg IV over 30-60 minutes** may be administered.
- **Lidocaine 1 mg/kg IV** is a final option for a wide QRS and should only be used in consultation with a pediatric cardiologist. A 12-lead ECG should be obtained prior to and after conversion from SVT to normal sinus rhythm.

Paediatric formulas and drug doses for arrest and peri-arrest scenarios**WET FLAG FORMULA**

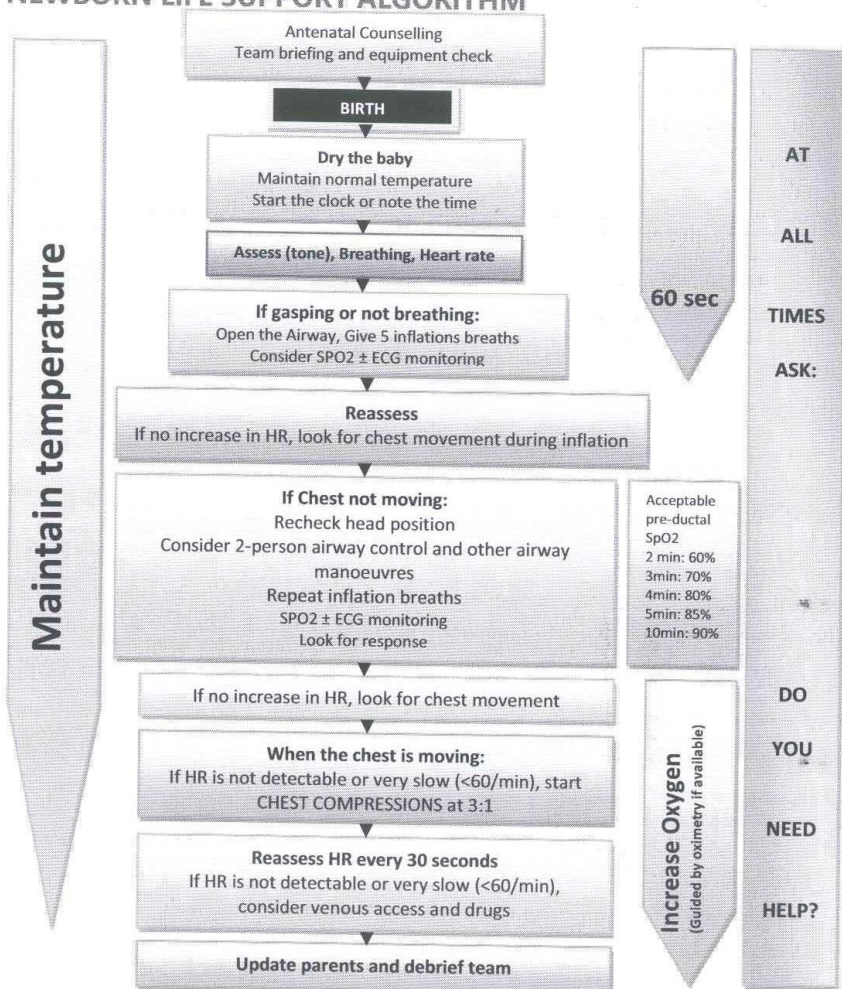
Weight	<ul style="list-style-type: none"> • 1–12 months = $(0.5 \times \text{age months}) + 4 = \frac{1}{2}\text{age in months} + 4$ • 1–5 years = $(2 \times \text{age years}) + 8$ • 6–12 years = $(3 \times \text{age years}) + 7$
Electricity	<ul style="list-style-type: none"> • Defibrillation 4 J/kg (single shocks) • Cardioversion 1 J/kg then 2 J/kg then amiodarone and repeat
Tube: Endotracheal tube (ETT)	<ul style="list-style-type: none"> • Length: <ul style="list-style-type: none"> ○ Oral ETT = $\text{Age}/2 + 12$ and Nasal ETT = $\text{Age}/2 + 15$ • Children aged over 1 year: <ul style="list-style-type: none"> ○ Diameter in mm: $\text{Age}/4 + 4$ • Children under 1 year the following sizes should be used: <ul style="list-style-type: none"> ○ Neonates under 3 kg – size 3.0 or 3.5 mm ○ Age 6 months – size 4.0 mm ○ Age 1 year – size 4.5 mm
Fluids	<ul style="list-style-type: none"> • Boluses are 20ml/kg of Normal saline • Trauma: 10ml/kg (Blood: 10ml/kg) • DKA: 10 ml/kg. • Burns: $\% \text{ burn} \times \text{weight} \times 4$ ($\frac{1}{2}$ given in first 8 h, $\frac{1}{2}$ given over next 16 h)
Lorazepam	0.1mg/kg IV/IO
Adrenaline	<ul style="list-style-type: none"> • Cardiac arrest: 0.1 mg/kg IV/ IO (0.1 ml/kg of 1: 10,000) • Anaphylaxis: <ul style="list-style-type: none"> ○ Age >12 years 0.5 mg IM. ○ Age 6–12 years 0.3 mg IM. ○ Age <6 years 0.15 mg IM. • Croup: 0.5ml/kg (max. 5 ml) of 1:1000 nebulized
Glucose	<ul style="list-style-type: none"> • Dextrose 10% 2 ml/kg

DRUG DOSES AND INDICATIONS

Amiodarone	<ul style="list-style-type: none"> • Shockable cardiac arrest rhythms (VF/VT) 5 mg/kg IV (after 3rd shock) • Pulsed VT 5 mg/kg IV
Atropine	<ul style="list-style-type: none"> • 0.02 mg/kg (minimum dose 0.1 mg, maximum 0.6 mg) • Use pre-intubation or for bradycardia secondary to vagal stimulation. Not recommended in cardiac arrest.
Budesonide	<ul style="list-style-type: none"> • Croup: 2 mg nebulizer
Ceftriaxone or cefotaxime	<ul style="list-style-type: none"> • Meningitis: 80 mg/kg IV (avoid ceftriaxone in neonates)
Dexamethasone	<ul style="list-style-type: none"> • Croup: 0.15–0.6 mg/kg
CPR	<ul style="list-style-type: none"> • Ratio 15:2 (5 rescue breaths first) • Rate: 100–120 per min • Hand positioning: Lower $\frac{1}{2}$ of sternum (locate as 1 finger-breadth above xiphisternum) • Depth of compression: At least $\frac{1}{3}$ depth of chest • Technique: Infant 2 fingers (or encircling technique with 2 thumbs). Child 1 or 2 hands.

II. NEONATAL RESUSCITATION

NEWBORN LIFE SUPPORT ALGORITHM



Many maternity care providers continue to clamp the umbilical cord immediately after an uncomplicated vaginal birth, even though the significant neonatal benefits of delayed cord clamping (usually defined as 2 to 3 minutes/ others 3-5 minutes after birth) are now well known.

APGAR SCORE

Check for:

- Appearance
- Pulse
- Grimace
- Activity
- Respirations

CHAPTER 3. CHOKING CHILD**GENERAL SIGNS OF CHOKING**

- Witnessed episode
- Coughing or choking
- Sudden onset
- Recent history of playing with or eating small objects

SEVERE AIRWAY OBSTRUCTION:**Ineffective coughing**

- Unable to vocalise
- Quiet or silent cough
- Unable to breathe
- Cyanosis
- Decreasing level of consciousness

MILD AIRWAY OBSTRUCTION:**Effective cough**

- Crying or verbal response to questions
- Loud cough
- Able to take a breath before coughing
- Fully responsive

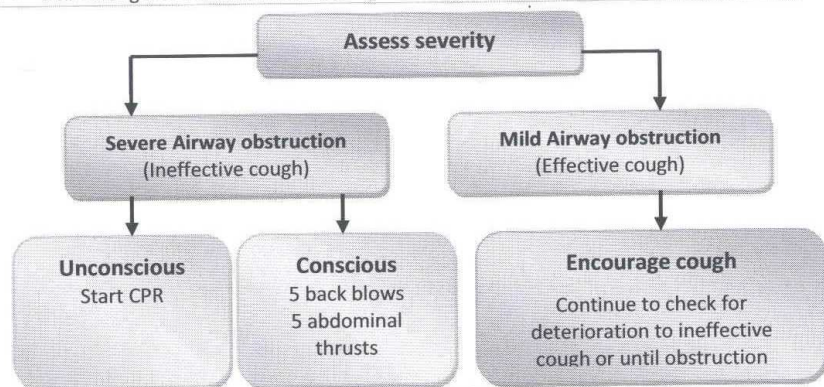


Figure 5.3. 2. Paediatric choking algorithm

Infants < 1 year: Chest thrusts and infants >1 year abdominal thrusts.

Unconscious child with choking

- If the choking child is, or becomes, unconscious place him on a firm, flat surface.
- Call out, or send, for help if it is still not available.
- Do not leave the child at this stage.

Airway opening:

- Open the mouth and look for any obvious object.
- If one is seen, make an attempt to remove it with a single finger sweep.

Do not attempt blind or repeated finger sweeps – these can push the object more deeply into the pharynx and cause injury.

Rescue breaths:

- Open the airway and attempt 5 rescue breaths.
- Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.
- **Attempt 5 rescue breaths** and if there is no response, proceed immediately to chest compression regardless of whether the breaths are successful.

CHAPTER 4. APPROACH TO PEDIATRIC TRAUMA

I. MAJOR TRAUMA IN CHILDREN

INITIAL ACTIONS

- Always start with the primary survey
- ADDRESS any problems found before moving on with your evaluation
- Log roll the patient and get them off the backboard when able
- Secondary survey with FAST exam and x-rays
- If the patient begins to deteriorate, reassess the patient and restart your evaluation with the ABC's. Remember this is likely a very terrified child and this can alter your exam.
- When able, always include family members to help calm and comfort the child.

1. PRIMARY SURVEY

- The **initial** assessment and survey is not significantly different from adults. However, children have a unique physiologic response to trauma, creating challenges in their management.
- Start with the primary survey as with adult patients. Always have a consistent, systematic approach in the evaluation of a patient with traumatic injuries (i.e. Advanced Trauma Life Support or ATLS).
 - Airway maintenance with cervical spine protection
 - Breathing and ventilation
 - Circulation with haemorrhage control
 - Disability (neurological status)
 - Exposure and environmental control (completely undress the patient but avoid hypothermia)
 - F – Family

A. AIRWAY

- The first priority is determining if the airway is open and patent.
- Always maintain C-spine immobilization
- Is the child speaking? If they are non-verbal, are they crying?
- This is a frightening experience for most young children so be wary of the silent child
- Pediatric airways have similar complications to adult airways that may lead to loss of a patent airway and a difficult intubation.
- Foreign body in airway/ Facial fractures and bleeding/
- Tracheal injuries
- Expanding hematomas
- Depressed level of consciousness and inability to protect airway
- If a child does not have a patent airway or is at risk of losing their airway, act quickly:
 - Use **simple airway manipulations** (jaw thrust, nasopharyngeal airway, or an oropharyngeal airway) or bag-valve mask ventilations if these seem ineffective.
 - **Rapid sequence intubation (RSI)** may be required to stabilize a lost airway or protect a tenuous one
 - Children with significant airway edema, abnormal anatomy, foreign body, or significant maxillofacial injuries may require **cricothyroidotomy** (surgical or needle)
 - Generally, children less than 8-10 years old are not candidates for cricothyroidotomy and may require **transtracheal ventilation (jet ventilation)**.

- The pediatric airway has both anatomic and physiologic differences that set it apart from an adult airway.
 - *Prominent occiput* – forces flexion of the neck in the supine position potentially obstructing the airway making intubation difficult
 - *Larger tongue*
 - *Larger adenoid tissue*
 - *Floppy, omega shaped epiglottis*
 - *Larynx is more cephalad and anterior*
 - *Cricoid ring, not the vocal cords, is the narrowest portion of the airway*
 - *Shorter tracheal length*

B. BREATHING

- Once you have evaluated and stabilized the airway, it's time to evaluate the patient's breathing. As with adults, you will need to **look, feel, and listen** to the chest in children.
- **Inspect** – Look for tracheal deviation, accessory muscle use, chest wall injury, and paradoxical breathing (chest wall moves inward with inspiration and outward with expiration)
- **Palpate**– Feel for crepitus and point tenderness, paradoxical movements with observed paradoxical breathing, or percuss for hyper-resonance or dullness
- **Auscultate** – Listen for signs of upper airway obstruction (**stridor**) or signs of pneumothorax (**absent breath sounds**) or hemothorax (**diminished breath sounds**)
- Auscultation, especially in small children, is the least reliable evaluation of breathing. In small children, you can hear lung sounds from the adjacent lung even if there is a pneumothorax in the lung that is auscultated.
- If a child has a **patent airway** but your evaluation reveals **poor breathing**, ventilating the child is your next priority:
 - **Bag-valve mask (BVM)** ventilation is a temporizing method to ventilate a child, and a very important skill! BMV ventilated until an advanced airway can be placed.
 - **An oral airway** can greatly assist ventilation in children with macroglossia or any other upper airway obstruction.

C. CIRCULATION

- Children have a remarkable ability to maintain their blood pressure even with significant volume loss (25-30% blood volume loss).
- *Since children compensate so well (compensated shock), the blood pressure is not as accurate a reflection of circulatory status as it is in adults.*
- Feel for central and peripheral pulses (tachycardia is common)
- Skin color/ Capillary refill
- Though it is less helpful during the initial survey, **urine output** is one of the best indicators of volume status (**>1-2 ml/kg/h**)
- **Hemorrhagic shock** is the most common etiology of shock in the setting of a traumatic injury. Intravenous access is essential: obtain **2 large bore intravenous catheters**
- If peripheral intravenous access is difficult or taking too much time, rapid placement of an intraosseous (IO) needle can be life-saving
- Initially give **10ml/kg of isotonic fluids** (typically normal saline)
- Fluids should be given rapidly using either a pressure bag or via the 3-way stopcock “pull-push” method. If a child has not had an adequate response after 2 boluses, consider giving **10ml/kg of packed red blood cells**. Continue to fluid resuscitate the child during the ongoing evaluation and treatment of specific injuries.

D. DISABILITY: NEUROLOGIC ASSESSMENT

- Similar to adults, the disability examinations should rapidly assess a child for neurologic deficits.
- The American Heart Association Pediatric Advanced Life Support (PALS) program recommends the use of the mnemonic AVPU in children as well.
 - Alert** – fully alert
 - Voice** – responds to voice
 - Pain** – responds to pain
 - Unresponsive** – is completely unresponsive
- A quick pupillary examination is typically done at this time as well. You are assessing for size, symmetry, and reactivity. Unresponsive and dilated pupils are worrisome for uncal herniation and emergent intervention will be needed.
- PALS also recommends the use of the validated Modified Glasgow Coma Scale for Infants and Children to quickly evaluate mental status. The best possible score is 15 and the lowest is 3.

Category	Child	Infant	Score
Eye Opening	Spontaneous	Spontaneous	4
	To speech	To speech	3
	To pain only	To pain only	2
	No response	No response	1
Best Verbal Response	Oriented, appropriate	Coos and babbles	5
	Confused	Irritable cries	4
	Inappropriate words	Cries to pain	3
	Incomprehensible sounds	Moans to pain	2
	No response	No response	1
Best Motor Response (The most important part if intubated, unconscious, or preverbal)	Obeys commands	Moves spontaneously and purposefully	6
	Localizes to painful stimulus	Withdraws to touch	5
	Withdraws to pain	Withdraws to pain	4
	Flexion in response to pain	Abnormal flexion posture to pain	3
	Extension in response to pain	Abnormal extension posture to pain	2
	No response	No response	1

- A rapid bedside glucose should be checked as well.** Children have smaller glycogen stores as well as a higher metabolic demand and can become hypoglycemic. Hypoglycemia will have a profound effect on a child's mental status and will require emergent interventions (typically a bolus of intravenous dextrose).
- Finally, **logroll the child** while maintaining cervical spine immobilization and palpate the spine for tenderness and step-offs. Remove the backboard at this time as well. Logrolling is a carefully orchestrated maneuver in an effort to maintain inline cervical spine immobilization.

E. ENVIRONMENT/EXPOSURE

- All children must be fully undressed and examined thoroughly for hidden injuries.
- The metabolic needs of children are increased by hypothermia and maintenance of normothermia is paramount.
- This is accomplished with the use of **warm blankets, warmed intravenous fluids, and a warm ambient temperature in the trauma bay.**

F. FAMILY

- The emergency department trauma bay can be loud and frightening for young children, impacting the assessment of a child. Allowing family members to comfort and guide child during the evaluation can provide a more accurate evaluation.
- A child life specialist can be invaluable if a family member cannot be present.
- It has been shown that family presence during a resuscitation is beneficial to the grieving process as well. However, one specific person (child life, chaplain, social worker) should be the point person who can answer all of their questions during this experience.
- Surprisingly, the largest barrier to implementing this practice has been due to the medical providers.

2. SECONDARY SURVEY

- The secondary survey is a **head to toe examination** that only begins once all problems identified on the primary survey have been addressed and the child is stable.

HISTORY

- A focused history is best obtained using the **AMPLE mnemonic**:
 - **A** = Allergies
 - **M** = Medications
 - **P** = Past medical history
 - **L** = Last meal
 - **E** = Environments and events associated with injury

PHYSICAL EXAM**1. HEAD TRAUMA**

- Head injury is the leading cause of death and disability in children.
 - The relative size of a child's head compared to their trunk is larger which places more torque on the cervical spine.
 - Children are also more susceptible to shear injury because their brains are less myelinated. Severe head injury is the leading cause of death in children 1-18 years.
 - **Always consider non-accidental injury when evaluating a child with head trauma.**
- As a result, the head exam must be thorough and systematic in children.
- Inspect and palpate the skull for **step-offs** or **lacerations**
- **Battle's sign** (bruising behind the ear), **raccoon eyes** (periorbital bruising without swelling), and/or **hemotympanum** (blood behind the ear drum) are worrisome for a basilar skull fracture
- **Scalp hematomas**, particularly non-frontal are worrisome
- Feel for the **anterior fontanelle** in a child under 1 year
- An age appropriate neurologic examination to assess motor and sensory function as well as cranial nerves (a caregiver may notice a subtle abnormality).
- Attempt to limit radiation and unnecessary head CT scans by **NICE HEAD INJURY GUIDELINES** (see Adult head Injury section).
- The dental exam can easily be overlooked but should be performed especially looking for injuries that may damage underlying permanent teeth (e.g. **impacted tooth**)
- **Neck and Spine Trauma**
 - Cervical spine injuries in children vary from adults due to anatomic differences.
 - *Children have relatively larger heads*
 - *Their ligaments and joint capsules are more lax*
 - *Their facet joints are more horizontal*
 - *Their vertebral bodies are wedge shaped*

- Always assume a child has had a C-spine injury until proven otherwise.
- Most patients will arrive to the ED with pre-hospital C-spine immobilization.
- Maintain immobilization until an injury has been ruled-out.
- If a child is too small for a cervical collar, sandbags on either side of their head and tape are an alternative. While maintaining C-spine immobilization with a second provider, palpate for midline deformities or tenderness along C-spine. Thorough motor and sensory examination (isolated sensory complaints more common)
- Many children are asymptomatic or too uncooperative to provide an adequate examination (Use family and child life specialist liberally)
- Use NICE GUIDELINES as per adult section.

2. CHEST TRAUMA

- Children have thoracic anatomic and physiologic differences from adults
 - *Compliant chest wall*
 - *Fewer rib fractures*
 - *Mediastinum is more mobile*
- Compliance can mask underlying injuries and minimize external signs of trauma
- Though increased compliance leads to fewer rib fractures, it also leads to increased **pulmonary contusion**
- The physical exam evaluating the chest is similar in children compared to adults.
 - **Inspection:** nasal flaring, chest wall injuries, bruising, seat belt sign (shoulder belt), paradoxical chest wall movement
 - **Palpation:** crepitus and/or tenderness
 - **Auscultation:** muffled heart sounds, abnormal lung sounds (absent, muffled); *least reliable finding of the three*
- Isolated thoracic injury is uncommon in children. It is more likely to result with a significant injury causing concomitant injuries. Children will have injuries similar to adults.
 - **Pneumothorax / Hemothorax**
 - **Pulmonary contusion**
 - Responsible for ~ 10% of all pediatric trauma admissions
 - Mild to severe hypoxia depending on the extent of contused lung
 - Always be vigilant because it can worsen over time as contusion evolves
 - CXR findings may lag behind injury but if abnormal, represents a significant injury
 - **Flail chest:** Results from two or more fractures in contiguous ribs.
 - **Rib fractures**
 - **Traumatic asphyxiation**
 - Due to increased compliance of the pediatric chest wall. Occurs after direct compression of the chest and deep inspiration against a closed glottis with a crush injury. This increases pressure in the superior and inferior vena cava and leads to facial/neck hemorrhage, cyanosis, and facial swelling.
 - Treat by addressing associated injuries and elevating the head of the bed.
 - **Commotio cordis:**
 - Almost solely a pediatric traumatic injury. This is a combination of direct anterior chest injury leading to ventricular fibrillation and sudden cardiac death.
 - Treatment consists of rapid recognition and use of an automated external defibrillator by by-standers or first responders.

3. ABDOMINAL TRAUMA

- Children have specific anatomic differences that protect them from some injuries but place them at greater risk for others:
 - *Larger solid organs*
 - *Less subcutaneous fat*
 - *Less protective abdominal wall musculature*
 - *Larger kidneys*
 - *Flexible rib cage allowing for excursion of the chest wall and abdominal organ compression*
- The abdominal exam for children consists of:
 - **Inspection:** vital signs (hypotension, tachycardia, or tachypnea are worrisome), completely undress children, bruising (seat belt sign, periumbilical, flank), or tire tracks may be seen.
 - **Auscultation:** hypoactive bowel sounds
 - **Palpation:** point tenderness, rebound, guarding
 - **Percussion:** diffuse dullness is a sign of peritoneal injury
 - **FAST or Focuses Assessment Sonography in Trauma** is a quick and non-invasive ultrasonographic evaluation of the abdomen looking for free fluid.
 - 4 specific areas are visualized: **Morrison's Pouch (hepato-renal), perisplenic (spleno-renal), bladder, and subxiphoid cardiac view.**

4. PELVIC TRAUMA

- Pelvic injuries are uncommon in children.
- Children should still be inspected and palpated for signs of pain or pelvic instability.
- If there is concern, pelvic films should still be ordered.
- If there is concern for instability and a pelvic fracture, compression with a wrapped sheet or a pelvic binder should still be placed.

5. GENITAL, PERINEAL, AND RECTAL TRAUMA

- Any signs of genital hematomas, blood at the urethral meatus, or lacerations should be evaluated further.
- If there is concern for blood, a rectal exam should still be performed.
- Often times, visualized rectal tone (anal wink) is sufficient unless neurologic injury (spinal shock) is a concern and then a digital rectal exam should be performed.

6. MUSCULOSKELETAL TRAUMA

- A thorough extremity exam is always needed. Evaluate the neurovascular status.
- Patients with a gross deformity or point tenderness will need x-rays to evaluate for fracture. Splint deformed extremities to help prevent further injury and alleviate pain.

DIFFERENTIAL DIAGNOSIS

- The most life-threatening diagnoses that will need emergent intervention include:
 - *Traumatic brain injury with increased ICP and impending herniation*
 - *Airway obstruction*
 - *Respiratory failure/ Large pulmonary contusion*
 - *Tension pneumothorax/ Massive hemothorax*
 - *Comotio Cordis*
 - *Blunt abdominal trauma with active hemorrhage*

II. BURNS IN CHILDREN

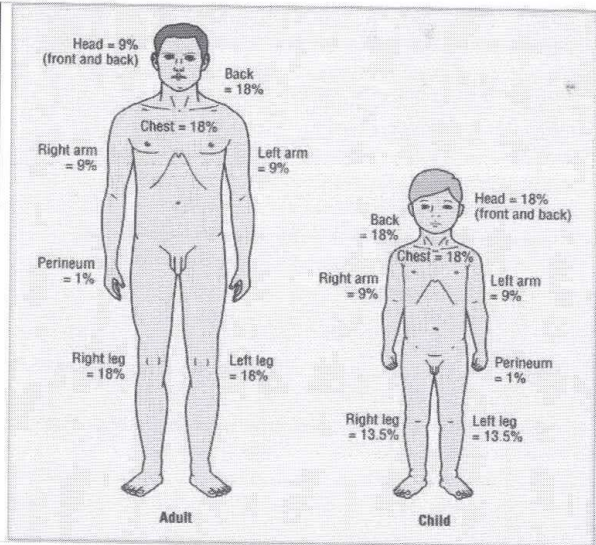
- The same principles apply for the management of burns in children as in adults.
- The possibility of non-accidental injury** must always be considered in a child with burns and appropriate safeguarding action taken.
- Suspicious patterns of burn include **cigarette burns**, **immersion-type injuries** (glove and stocking pattern or buttocks), or **burns to the dorsum of the hands**.
- Scalds from pulling hot drinks off surfaces are common accidental injuries.
- Advice should be given about how to prevent future similar injuries occurring.
- The history of the presentation may raise the suspicion of non-accidental injury:
 - Delay in seeking medical attention.
 - The details of the mechanism of injury are implausible, change over time, or are inconsistent with the developmental stage of the child.
 - Lack of concern by the person accompanying the child.
 - Abnormal behaviour or demeanour by the child e.g. withdrawn, avoiding eye contact. This should be observed in the context of the child's background – for example it is usual to avoid eye contact in some Pacific cultures.
 - Direct disclosure by the child that the injury was deliberately inflicted.

CALCULATING THE SIZE OF THE BURN

- Assessing the % of BSA burnt can be done using several different techniques:
 - Infant rule of 5's
 - Lund-Browder charts
 - Patient's palm size (including the fingers) = approximately 1% body surface area.
- Size should only include those burns which are partial or full thickness; superficial burns are excluded from the estimation. Accurate estimation is important for managing fluid resuscitation and communicating with a burns centre.

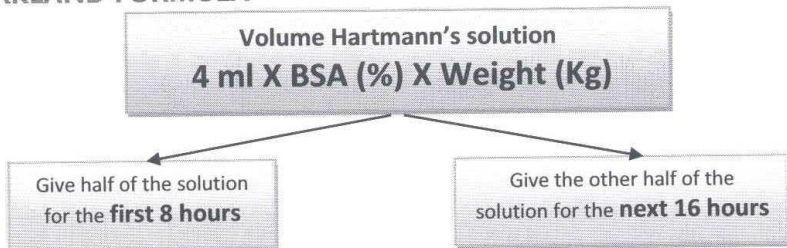
INFANT RULE OF 5' S

Body area	Infant rule of 5's
Head	20%
Each arm	10%
Each leg	20%
Front of trunk	10%
Back of trunk	10%



MANAGEMENT OF BURNS IN CHILDREN

- The management of burns in children is the same as in adults.
- Children with burns >10% body surface area require intravenous fluids.
- Fluid requirements are:
 - Calculated using the Parkland formula.
 - Maintenance fluid requirements will also need adding to the fluid calculation.

PARKLAND FORMULA**TRANSFERRAL CRITERIA TO A BURN CENTRE**

- *Patients requiring burn shock resuscitation.*
- *Burns > 5 % TBSA in a Child*
- *Full thickness burns > 5% TBSA*
- *Burns of face, hands, feet, perineum, genitalia, and major joints*
- *Circumferential burns*
- *Chemical or electrical burns*
- *Burns in the presence of major trauma or significant co-morbidity*
- *Burns in the very young patient*
- *Suspicion of Non-Accidental Injury*

DIFFERENCES BETWEEN CHILDREN AND ADULTS SUMMARISED BY SEVEN S'S:

- **Softer skeleton:** bones tend to bend rather than break. Children may have significant internal injuries despite an intact overlying skeleton. The identification of skull or rib fractures suggests the transfer of a massive amount of energy and underlying organ injuries should be suspected.
- **Surface area:** the ratio of a child's body surface area to volume is greater than an adult, resulting in faster temperature loss and the potential for hypothermia to develop quickly.
- **Size:** the size of a child affects the mechanism of injury and they are more likely to suffer multisystem trauma, and/or a higher force per unit area.
- **Shape:** children have bigger heads proportional to their body resulting in a higher frequency of blunt brain injuries.
- **Sequelae:** trauma in children may have an effect on their subsequent growth and development. The child has to recover from the effects of the traumatic event and continue the normal growing process, consequently long-term complications need to be considered from the outset.
- **Psychology (pPsychology):** children often regress by a stage when ill or injured. This can result in communication difficulties and involvement of the parents is paramount.
- **Stuff (Equipment):** different size equipment is needed and calculations for drug doses are required. An electronic medical application, length-based resuscitation tape (e.g. Broselow tape), or specific age-based resuscitation draws are useful for this.

CHAPTER 5. APPROACH TO PEDIATRIC SHOCK

I. THE SHOCKED CHILD

INTRODUCTION

- Shock is a term used to describe inadequate oxygen delivery to the tissues that cannot keep up with metabolic demand. This creates a state of hypoperfusion.
- It may be uncompensated, meaning there is hypotension and inability to maintain normal perfusion, or it may be compensated, meaning that blood pressure and perfusion are maintained for the time being.
- There are several different types of shock (below) and shock is often thought of as being "warm" or "cold."
- The intravascular volume of an infant is approximately **80 ml/kg**.
- In older children, the intravascular volume is approximately **70 ml/kg**.
- Dehydration in itself does not cause death, but shock does. Shock can occur with losses of **20 ml/kg** from the intravascular space, while clinical dehydration is only evident after total losses of greater than **25 ml/kg**.
- The maintenance fluid requirements for well, normal children are summarized in the table below:

Body weight	Daily fluid requirement	Hourly fluid requirement
First 10 kg	100 ml/kg	4 ml/kg
Second 10 kg	50 ml/kg	2 ml/kg
Subsequent kg	20 ml/kg	1 ml/kg

- Generally speaking, a child with clinical signs of dehydration but no evidence of shock can be assumed to be **5% dehydrated**. 5% dehydration implies that the body has lost 5 g per 100 g body weight i.e. **50 ml/kg of fluid**.
- If shock is also present that **10%** dehydration of greater can be assumed to have occurred.

CLINICAL FEATURES OF DEHYDRATION AND SHOCK

DEHYDRATION (5%)	CLINICAL SHOCK (10%)
<ul style="list-style-type: none"> Appears 'unwell' Normal HR or tachycardia Normal RR or tachypnoea Normal peripheral pulses Normal or mildly prolonged CRT Normal blood pressure Warm extremities Decreased urine output Reduced skin turgor Sunken eyes Depressed fontanelle Dry mucous membranes 	<ul style="list-style-type: none"> Pale, lethargic, mottled Tachycardia Tachypnoea Weak peripheral pulses Prolonged CRT Hypotension Cold extremities Decreased urine output Decreased level of consciousness

- **INITIAL ACTIONS**

- **Primary Survey:**

- **ABC's** are the critical first step in a patient with shock!
- Place the child on the **monitor, pulse oximeter, and obtain blood pressure.**
- **A&B:** Start supplemental oxygen and consider **early intubation** if the child will require ventilatory assistance, significant help with oxygenation, or airway protection.
- **C:** Obtain IV/IO access; give a **20 ml/kg bolus** of IV crystalloid.
 - This may be repeated twice up to a total fluid administration of **60 ml/kg.**
 - If the child remains in shock, this is considered refractory shock and it would then be prudent to consider adding **vasopressor support**, often in the form of **Norepinephrine or Dopamine.**
 - If a child has risk factors for adrenal insufficiency, consider administering stress-dose steroids as adrenal insufficiency can also lead to a refractory shock state.
 - If the child is suffering from hemorrhagic shock, **blood should be administered** after the initial crystalloid bolus and the site of hemorrhage should be managed appropriately.
- THEN identify the type of shock, which may not always be easy in mixed shock states (see Differential Diagnosis).

CARDIOGENIC SHOCK

- It is a special type of shock in which there is failure of the pump due to malformation, overload, obstruction, or non-perfusing rhythm. Fluid may still be given in this instance but at a lower bolus (**5-10 ml/kg**) and over a longer period of time (**up to 20 minutes**) to prevent exacerbation of the failure state and worsening pulmonary edema. Closely monitor fluid and respiratory status during fluid administration in this instance.
- If suspicious for a **ductal-dependent cardiac lesion or anomaly**, which can cause an obstructive shock picture with cardiogenic shock, you should also consider administering **prostaglandin** in this instance to open the ductus arteriosus which can ease the amount of vascular congestion and fluid backing up into the lungs.
- Discuss with pediatric cardiologist and/or pediatric cardiothoracic surgeon.

- **GOALS FOR RESUSCITATION SHOULD INCLUDE:**

- **Blood pressure** (*systolic pressure at least fifth percentile for age: 60 mmHg <1 month of age, 70 mmHg + [2 x age in years] in children 1 month to 10 years of age, 90 mmHg in children 10 years of age or older*)
- **Quality of central and peripheral pulses** (*strong, distal pulses equal to central pulses*)
- **Skin perfusion** (*warm, with capillary refill <2 seconds*)
- **Mental status** (*normal mental status*)
- **Urine output** (*≥1 mL/kg per hour, once effective circulating volume is restored*)
- **Clearance of lactate** (*hope to see down trending and preferably cut in half after initial resuscitation*)

1. HYPOVOLEMIC SHOCK

- This is the most common cause of shock worldwide in infants, most often secondary to diarrhea (Other examples include **blood loss, vomiting, heat stroke, or burns**).
- It is important to realize the stages of shock, especially in children who can compensate for a larger percentage of losses than adults and then rapidly decompensate.

2. DISTRIBUTIVE SHOCK

- Distributive shock often results from vasodilation and a decrease in systemic vascular resistance. It is associated with normal to increased cardiac output.
- Given the vasodilation, the extremities are warm, making this an example of "warm shock."
- **CAUSES OF DISTRIBUTIVE SHOCK INCLUDE:**
 - **Sepsis**
 - Most common etiology in children
 - Infection causes significant vasodilation
 - Think about in a child with fever and other signs of infection
 - **Anaphylaxis**
 - Causes profound vasodilation secondary to an IgE-mediated immediate hypersensitivity reaction
 - Think about in a child with wheezing, urticaria, angioedema, or stridor
 - **Neurogenic**
 - Spinal cord injury resulting in loss of sympathetic tone
 - This results in vasodilation as well as bradycardia
 - Think about in trauma patients with neurological deficits and paradoxical bradycardia in the setting of hypotension

3. CARDIOGENIC SHOCK

- Cardiogenic shock results from pump failure and depressed cardiac output. This decreased cardiac output results in cool extremities, another example of "cold shock."
- The most common causes of cardiogenic shock in children are as follows:
 - **Structural Disorders** – often present a picture of obstructive shock
 - Hypoplastic left heart syndrome, tetralogy of Fallot, coarctation of the aorta and other structural disorders can result in systolic heart failure
 - Think about in children with hepatomegaly, signs of pulmonary edema, JVD, or murmur
 - **Cardiomyopathies**
 - **Infections** such as myocarditis, familial causes such as hypertrophic obstructive cardiomyopathy.
 - **Infiltrative causes** such as hemochromatosis can cause myocardial dysfunction and failure.
 - Think about in children with recent infection, murmur, chest pain, or signs of heart failure.
 - **Arrhythmias**
 - Prolonged SVT or ventricular dysrhythmias can cause substantial decrease in stroke volume and thus cardiac output, also leading to failure.
- **DISPOSITION:**
 - Most patients who present with shock will require admission to a pediatric ICU for close monitoring, frequent reassessment, and further management.
 - Early consultation with an intensivist is recommended and you may need to contact other specialists including surgeons in the case of trauma or cardiac defect.

II. PAEDIATRIC GASTROENTERITIS

DEFINITION

- **Vomiting:** A forceful ejection of stomach contents up to and out of the mouth
- **Diarrhoea:** Passage of liquid or watery stools. In most cases there is an associated increase in frequency and volume. Gastroenteritis should therefore be suspected if there is a sudden change in stool consistency and/or vomiting.

CAUSATIVE ORGANISMS

- Artistic impression of the highly infectious **rotavirus**
- There is a positive culture in 45-75% of stool samples.
- **Viruses (30-57%)**
 - Rotavirus most common
 - Norovirus and Adenovirus also common
- **Bacteria (6-14%)**
 - Less common: Campylobacter, Salmonella and Shigella typically isolated
- **Protozoa (~1%):** Cryptosporidium most commonly isolated

DIFFERENTIAL DIAGNOSES

- Consider the following as potential diagnoses:
 - **Non-enteric infections:** Meningitis, Septicaemia, UTI, Pneumonia
 - **Non-infective gastroenterological conditions:** IBS, coeliac disease, malabsorption, overflow constipation
 - **Acute surgical abdominal conditions:** Appendicitis, volvulus, intussusception
 - **Antibiotic associated diarrhea:** including Clostridium Difficile
- Vomiting lasting more than 24 hours without diarrhoea should trigger consideration of an alternative diagnosis.
- **CLINICAL FEATURES OF DEHYDRATION AND SHOCK : see above**

RISK STRATIFICATION

- **The following children are at increased risk of dehydration:**
 - Young age (<1 year of age)
 - Infants with low birth weight
 - Those with signs of malnutrition
 - Frequent symptoms (>5 diarrhoeal stools or >2 vomits within the previous 24 hours)
 - Those who are not offered supplementary fluids or stopped breastfeeding prior to presentation

ASSESSMENT OF DEHYDRATION/NICE CG84 April 2009

- **Hypertonic dehydration (Na>150 mmol/L) should be suspected if:**
 - Child <6 months' old
 - CNS dysfunction
 - Jitteriness
 - Hypertonia, hyper-reflexia
 - Coma, convulsions

INVESTIGATIONS

- **Stool Culture**
 - The majority of cases of paediatric gastroenteritis are viral and even cases of bacterial or protozoal infection are generally self-limiting.

- Therefore, NICE CG84 suggests the following:
 - **Stool microbiology, culture and sensitivity (MC&S) should be performed if:**
 - *Septicaemia is suspected*
 - *There is blood and/or mucous in the stool*
 - *The child is immunocompromised*
 - **Consider stool MC&S if:**
 - *The child has recently been abroad*
 - *The diarrhoea has not improved by day 7*
 - *There is uncertainty regarding the diagnosis*

Laboratory Measures

- NICE guideline suggests the following:
 - *Do not routinely perform blood biochemistry in children with gastroenteritis*
 - Measure laboratory **U&Es & blood glucose** if:
 - IV fluid therapy is required
 - Hypernatraemic dehydration is suspected
 - Measure **venous blood acid–base status** if shock is suspected or confirmed
 - Take **blood cultures** if starting antibiotics

MANAGEMENT OF AGE IN THE ED

1. NO CLINICAL DEHYDRATION

- The aim is to **prevent** dehydration:
 - Discharge home from the ED
 - Reassure parents and carers that most cases can be safely managed at home
 - Provide verbal advice/ Continue breast feeds and other milk feeds
 - Encourage fluid intake/ Discourage fruit juices and carbonated drinks
 - If increased risk of dehydration, offer low osmolality **ORS** (i.e. Dioralyte®, Electrolade®) as a supplemental fluid. Seek advice from a healthcare professional if symptoms of dehydration develop. Advise on the typical duration of symptoms and to seek advice if they do not resolve within these timeframes.
- **Normal duration of symptoms**
 - Vomiting: 1–2 days, most stop within 3 days
 - Diarrhoea: 5–7 days, most stop within 2 weeks
- *In children who are not clinically dehydrated do not perform an in hospital 'fluid challenge'.*

2. CLINICAL DEHYDRATION (including Hypernatraemic)

A. ORAL REHYDRATION

- Continue to breastfeed (if applicable)
- Otherwise use low osmolality **ORS 50 ml/kg** (deficit replacement) plus maintenance fluid **over 4 hours**
- **Daily maintenance fluid** can be calculated using the child's body weight using the following formula: **Total Daily Maintenance (TDM):**
 - **100 ml/kg** for the 1st 10 kg
 - **50 ml/kg** for 2nd 10kg
 - **20 ml/kg** for every subsequent 1 kg
- ORS should be given often and in small amounts
- Actual volumes needed clearly depends upon requirement

- If ORS is refused by the child and **there are no 'red flags'**:
 - Consider other fluids (i.e. milk, water)
 - Avoid fruit juices and carbonated drinks
 - Consider NGT placement if the child is unable to drink and/or vomits persistently
 - Monitor response to oral fluids
- **If oral fluid is tolerated:**
 - Discharged home from ED
 - Reassure parents or carers that oral rehydration is usually possible
- **Provide verbal advice:**
 - Complete the remainder of the 4-hour fluid challenge at home
 - Administer the fluid in small, frequent amounts
 - Do not give other fluids unless advised
 - Do not give solid foods
 - Seek advice if the child refuses to drink or vomits persistently
- **Discuss with the paediatric team if:**
 - Electrolyte imbalance (inc. hypernatraemic dehydration)
 - An NGT is required
 - There is an indication for IV fluids

B. IV FLUID

- **IV fluids are only recommended in children with clinical dehydration if:**
 - *Evidence of deterioration during ORS therapy **and***
 - *Evidence of Red flags symptoms/signs **or***
 - *The child persistently vomits ORS*
- **If IV fluids are required:**
 - Obtain urgent expert advice on fluid management
 - Commence isotonic fluids for deficit correction and maintenance (0.9% saline or 0.9% saline and 5% glucose)
 - Rehydrate slowly (normally over 48 hours)
 - Monitor serum sodium level frequently
 - Aim for a reduction of less than 0.5 mmol/l per hour
- **During IV fluid therapy**
 - Gradually attempt to introduce oral fluids early
 - If tolerated, complete rehydration with oral fluid therapy
- **In cases of suspected hypernatraemic dehydration:**
 - Obtain baseline U&Es and blood glucose, Rapid correction can be dangerous
 - Ideally **oral rehydration** should be used.

3. CLINICAL SHOCK

- **ABC DEFG approach**
- Ensure patient airway, give high flow oxygen
- Obtain urgent IV access
- Measure baseline U&Es, blood glucose and venous blood gas
- Give a fluid bolus of **20 ml/kg 0.9% saline**
- **If remains shocked after bolus:**
 - Give a further bolus/ Consider other causes for shock
- **If remains shocked after 2nd bolus;** Consider discussion with paediatric ICU team

- **Once symptoms and signs of shock have resolved:**
 - Calculate daily maintenance requirement/ Add **100 ml/kg** deficit to fluid calculations
 - Commence isotonic fluids for deficit and maintenance
 - 0.9% saline or 0.9% saline and 5% glucose
 - Consider adding potassium to fluids once serum level is known
 - Monitor clinical and laboratory response to fluid therapy, adjust subsequent fluids as appropriate. Discuss with paediatric team.
- **Children who have hypernatraemic dehydration**
 - May be shocked at presentation/ Fluid resuscitation guidelines should be followed
 - Rehydration should be managed as per the guidance for those children who are clinically dehydrated.

WORKED EXAMPLE

- A 24 kg child responded to a 20 ml/kg fluid bolus and is no longer shocked. What is his initial hourly IV fluid requirement? What type of fluid would you prescribe?
- **Answer:**
 - Deficit = $100 \text{ ml/kg} \times 24 \text{ kg} = 2400 \text{ ml}$
 - Daily maintenance = $(100 \text{ ml/kg} \times 10 \text{ kg}) + (50 \text{ ml/kg} \times 10 \text{ kg}) + (20 \text{ ml/kg} \times 4 \text{ kg}) = 1580 \text{ ml}$
 - Hourly requirement = $2400 + 1580 \div 24 = 165 \text{ ml/hour}$
 - Start with **0.9% saline or 0.9% saline and 5% glucose**
 - Consider adding potassium once serum levels are known

4. ADDITIONAL THERAPIES

- The following therapies have been suggested in the management of paediatric gastroenteritis:
- **Antibiotics**
 - **Antibiotics should only be given in cases of:**
 - Suspected or confirmed septicaemia.
 - Extra-intestinal spread of bacterial infection.
 - Salmonella infection:
 - Children <6 months of age.
 - Malnourished or immunocompromised children.
 - Clostridium difficile-associated pseudomembranous enterocolitis, giardiasis, dysenteric shigellosis, dysenteric amoebiasis or cholera.
 - Specialist advice should be sought in children who have recently returned from abroad.
- **Anti-diarrheal agents**
 - NICE do not recommend their use in children with gastroenteritis.
- **Probiotics and Antiemetics:** No recommendations in NICE guidelines.

PUBLIC HEALTH CONSIDERATIONS

- **Parents and carers should be advised how to prevent spread of the infection:**
 - Washing hands in warm, soapy water after going to the toilet, changing nappies and preparing, serving or eating food
 - Towels used by the infected child should not be shared
 - The child should not return to school until asymptomatic for 48 hours
 - The child should not swim in a public pool until asymptomatic for 2 weeks
 - Notify and act on the advice of the public health authorities if you suspect an outbreak of gastroenteritis.

III. DKA - PAEDIATRIC**NICE Guideline NG18****RECOGNITION, REFERRAL AND DIAGNOSIS**

- If the plasma glucose level is above **11 mmol/litre** in a child or young person without known diabetes, and they have symptoms that suggest diabetic ketoacidosis (DKA), suspect DKA and immediately send them to a hospital with acute paediatric facilities.
- *Be aware that children and young people taking insulin for diabetes may develop DKA with normal blood glucose levels.*
- Suspect DKA **even if the blood glucose is normal** in a child or young person with known diabetes and any of the following:
 - *Nausea or vomiting*
 - *Abdominal pain*
 - *Hyperventilation*
 - *Dehydration*
 - *Reduced level of consciousness.*
- When DKA is suspected in a child or young person with known diabetes measure the **blood ketones** (beta-hydroxybutyrate), using a near-patient method if available. If the level is elevated, immediately send them to a hospital with acute paediatric facilities.
- When DKA is suspected in a child or young person with known diabetes and it is not possible to measure the blood ketones (beta-hydroxybutyrate) using a near-patient method, immediately send them to a hospital with acute paediatric facilities.
- When a child or young person with suspected or known DKA arrives at hospital, measure their:
 - *Capillary blood glucose*
 - *Capillary blood ketones (beta-hydroxybutyrate) if near-patient testing is available, or urine ketones if it is not*
 - *Capillary or venous pH and bicarbonate.*
- Diagnose DKA in children and young people with diabetes who have:
 - **Acidosis** (indicated by blood **pH below 7.3** or **plasma bicarbonate below 18 mmol/litre**) and
 - **Ketonaemia** (indicated by blood beta-hydroxybutyrate **above 3 mmol/litre**) or **ketonuria** (**++ and above** on the standard strip marking scale).
- **Mild to Moderate DKA: pH 7.1 and above**
- **Severe DKA: pH below 7.1.**

INITIAL MANAGEMENT

- Inform the responsible senior clinician once a diagnosis of DKA in a child or young person is made.
- When DKA is diagnosed in a child or young person in hospital, record their:
 - Level of consciousness
 - Vital signs (HR, BP, T^o, RR [look for kussmaul breathing])
 - History of nausea or vomiting and Clinical evidence of dehydration
 - Body weight.
- When DKA is diagnosed in a child or young person in hospital, measure and record the capillary or venous:
 - *pH and pCO₂*
 - *Plasma sodium, Potassium*

- *Urea and Creatinine*
- *Plasma bicarbonate*
- Children and young people with DKA should be cared for with one-to-one nursing either on a high-dependency unit (preferably a paediatric unit), or on a general paediatric ward, if:
 - *They are younger than 2 years or*
 - *They have severe DKA (indicated by a blood pH below 7.1).*
- Insert a **nasogastric tube** if reduced level of consciousness and vomiting, to **reduce the risk of aspiration**.
- Seek **urgent anaesthetic review** and discuss with a paediatric critical care specialist if a child cannot protect their airway because they have a **reduced level of consciousness**.
- **Discuss the use of inotropes** with a paediatric critical care specialist if **hypotensive shock**.
- Think about **sepsis** in a child or young person with DKA who has any of the following:
 - *Fever or hypothermia*
 - *Hypotension*
 - *Refractory acidosis*
 - *Lactic acidosis*.

FLUID AND INSULIN THERAPY

- Treat DKA with **oral fluids and subcutaneous insulin only** if the child or young person is **alert, not nauseated or vomiting, and not clinically dehydrated**.
- Treat DKA **with intravenous fluids and intravenous insulin** if the child or young person is **not alert, is nauseated or vomiting or is clinically dehydrated**.
- **Do not give** an intravenous fluid bolus to children and young people with mild or moderate DKA (indicated by a blood pH of **7.1 or above**).
- **Do not routinely give** an intravenous fluid bolus to a child or young person with severe DKA (indicated by a blood pH below 7.1).
- **Do not give** more than one intravenous fluid bolus of **10 ml/kg 0.9% sodium chloride** to a child or young person with severe DKA (indicated by a blood pH below 7.1) without discussion with the responsible senior paediatrician.
- In children and young people with DKA, calculate their **total fluid requirement for the first 48 hours** by adding the **estimated fluid deficit to the fluid maintenance requirement**.
- When calculating the fluid requirement for children and young people with DKA, assume:
 - **a 5% fluid deficit** in mild to moderate DKA (indicated by a blood pH of 7.1 or above)
 - **a 10% fluid deficit** in severe DKA (indicated by a blood pH below 7.1).
- Calculate the maintenance fluid requirement for children and young people with DKA using the following 'reduced volume' rules:
 - *if they weigh less than 10 kg, give 2 ml/kg/hour*
 - *if they weigh between 10 and 40 kg, give 1 ml/kg/hour*
 - *if they weigh more than 40 kg, give a fixed volume of 40 ml/hour.*
- These are lower than standard fluid maintenance volumes because large fluid volumes are **associated with an increased risk of cerebral oedema**.
- Aim to replace the fluid deficit evenly over the first 48 hours because faster rehydration is associated with an increased risk of cerebral oedema.
- **Use 0.9% sodium chloride without added glucose** for both rehydration and maintenance fluid in children and young people with DKA until the plasma glucose concentration is **below 14 mmol/litre**.

- Ensure that all fluids (except any initial bolus) administered to children and young people with DKA contain 40 mmol/litre potassium chloride, unless they have renal failure.
- If more than 20 ml/kg of fluid has been given by intravenous bolus to a child or young person with DKA, subtract any additional bolus volumes from the total fluid calculation for the 48-hour period.
- A 20 kg 6-year-old boy who has a pH of 7.15, who did not have a sodium chloride bolus, will require:
 - Deficit $5\% \times 20 \text{ kg} = 1000 \text{ mls}$ divide over 48 hours = 21 ml/hr plus
 - Maintenance 1ml/kg/hr = 20 ml/hr
 - Total = 41 ml/hour
- A 60 kg 16-year-old girl with a pH of 6.9, and who was given 30 ml/kg 0.9% sodium chloride for circulatory collapse will require:
 - Deficit $10\% \times 60 \text{ kg} = 6000 \text{ mls}$ minus 10ml/kg Resuscitation Fluid = - 600 ml divide over 48 hours = 113 ml/hr plus
 - Maintenance fixed rate = 40 ml/hr
 - Total = 153 ml/hour
- Do not give intravenous sodium bicarbonate to children and young people with DKA.
- Think about inserting a urinary catheter if it is not possible to accurately measure urine output for a child or young person with DKA.
- Do not give children and young people with DKA additional intravenous fluid to replace urinary losses.
- Start an intravenous insulin infusion 1–2 hours after beginning intravenous fluid therapy.
- When treating DKA with intravenous insulin, use a soluble insulin infusion at a dosage between 0.05 and 0.1 units/kg/hour. Do not give bolus doses of intravenous insulin.
- If a child or young person with DKA is using insulin pump therapy, disconnect the pump when starting intravenous insulin therapy.
- In discussion with a diabetes specialist, think about continuing subcutaneous basal insulin in a child or young person who was using a basal insulin before the onset of DKA.
- Change fluids to 0.9% sodium chloride with 5% glucose and 40 mmol/litre potassium chloride once the plasma glucose concentration falls below 14 mmol/litre.
- If during treatment for DKA plasma glucose falls below 6 mmol/litre:
 - Increase the glucose concentration of the intravenous fluid infusion, and
 - If there is persisting ketosis, continue to give insulin at least 0.05 units/kg/hour.
- If the blood beta-hydroxybutyrate level is not falling within 6–8 hours in a child or young person with DKA, think about increasing the insulin dosage to 0.1 units/kg/hour or greater.
- Think about stopping intravenous fluid therapy if ketosis is resolving, they are alert, and they can take oral fluids without nausea or vomiting.
- Do not change from intravenous insulin to subcutaneous insulin until ketosis is resolving, they are alert, and they can take oral fluids without nausea or vomiting.
- Start subcutaneous insulin at least 30 minutes before stopping intravenous insulin.
- For a child or young person with DKA who is using insulin pump therapy, restart the pump at least 60 minutes before stopping intravenous insulin. Change the insulin cartridge and infusion set, and insert the cannula into a new subcutaneous site.

MONITORING DURING THERAPY

- Monitor and record the following at least hourly in children and young people with DKA:
 - *Capillary blood glucose*
 - *Vital signs (heart rate, blood pressure, temperature, respiratory rate [look for Kussmaul breathing])*
 - *Fluid balance, with fluid input and output charts*
 - *Level of consciousness (using the modified Glasgow coma scale).*
- Monitor children and young people receiving intravenous therapy for DKA using **continuous electrocardiogram (ECG)** to detect signs of **hypokalaemia, including ST-segment depression and prominent U-waves.**
- At 2 hours after starting treatment, and then at least every 4 hours, carry out and record the results of the following blood tests in children and young people with DKA:
 - *Glucose (laboratory measurement)*
 - *Blood pH and pCO₂*
 - *Plasma sodium, potassium and urea*
 - *Beta-hydroxybutyrate.*
- Update the child and young person with DKA and their family members or carers (as appropriate) regularly about their progress.

COMPLICATIONS

• CEREBRAL OEDEMA

- Immediately assess children and young people with DKA for suspected cerebral oedema if they have any of these early manifestations:
 - *Headache*
 - *Agitation or irritability*
 - *Cushing Triad: Unexpected Bradycardia, Hypertension and Abnormalities of breathing pattern, for example respiratory pauses*
 - *Deterioration in level of consciousness*
 - *Oculomotor palsies*
 - *Pupillary inequality or dilatation.*
- If cerebral oedema is suspected in a child or young person with DKA, treat immediately with the most readily available of:
 - ***Mannitol 20%, 0.5–1 g/kg over 10–15 minutes or***
 - ***Hypertonic sodium chloride 2.7% or 3%, 2.5–5 ml/kg over 10–15 minutes.***
- After starting treatment for cerebral oedema with mannitol or hypertonic sodium chloride in a child or young person with DKA, immediately seek specialist advice on further management, including which care setting would be best.

• HYPOKALAEMIA

- If a child or young person with DKA develops hypokalaemia ($K < 3$ mmol/litre):
 - Think about ***temporarily suspending the insulin infusion***
 - Discuss hypokalaemia management urgently with a paediatric critical care specialist, because a central venous catheter is needed for intravenous administration of potassium solutions above 40 mmol/litre.

• VENOUS THROMBOEMBOLIC DISEASE

- Be aware of the increased risk of venous thromboembolism in children and young people with DKA, especially those with central venous catheters.

CHAPTER 6. THE UNCONSCIOUS CHILD

DIFFERENTIAL DIAGNOSIS

- Decreased consciousness is a non-specific sign with a wide differential diagnosis: **TIPS AEIOU**
 - Trauma
 - Intracranial infection
 - Poisoning
 - Shock: hypovolaemic, distributive and cardiogenic
 - Sepsis
 - Epilepsy
 - Raised intracranial pressure
 - Metabolic disease
 - Hypertension

AVPU SCORE

- **A: Alert**
- **V:** Responsive to **verbal** stimuli
- **P:** Responsive to **painful** stimuli
- **U: Unresponsive**

GENERAL QUESTIONS

- Exploring developmental milestones, past medical, travel, immunisation and family history including infant deaths further guides management. Non-accidental injury may be behind the cause of reduced consciousness, consider child protection issues.

HISTORY

- The key questions on presentation should explore prodromal events leading to decreased consciousness with reference to the wide differential diagnosis: any recent illness and length of symptoms:

Category	Symptoms
Shock	Abdominal pain, excessive diarrhoea and vomiting may suggest fluid loss or surgical cause.
Sepsis	Vomiting, headache, fever, rash and infectious exposure may suggest infection. However recent antibiotic use may mask classical presentations of meningitis in the early phase. A detailed history is recommended.
Trauma	Trauma may or may not be evident particularly in the case of a shaken baby . Inappropriate responses or inconsistencies and delays in seeking help arouse suspicion of non-accidental injury.
Intracranial	History of ear pain is suggestive of otitis media; ask about frontal headaches, facial pains and purulent nasal discharge which are suggestive of sinusitis. Intracranial extension can occur.
Epilepsy	There may be a family history or prior seizures or a history of neuro developmental delay.
Poisoning	No history may be given. Examination may give clues to potential source.
Raised intracranial pressure	If there is a past history of neuro-developmental problems, check whether a shunt has been inserted or if there is a history of hydrocephalus. Make enquiries regarding recent head injuries.
Metabolic	Recent weight loss, polydipsia or polyuria may suggest a metabolic cause. A family history should be sought including if any consanguinity which may suggest inborn errors of metabolism.
Hypertension	A review of medication history may give a clue to the cause.

INVESTIGATIONS – MONITORING

- The following should be monitored: **MOVER (Monitor-Oxygen-Vitals-ECG-Resus)**
- **Core Blood and Urine Tests**
 - **Bedside test: Capillary glucose** within 15 minutes of presentation.
 - **Blood gas:** in all cases
 - **Sepsis: Urinalysis, FBC, CRP, Blood Culture**
 - **Metabolic-specific cases:** VBG/ABG, Glucose, Urines Ketones, LFT, U&E, Serum Ammonia
 - **Overdose:** Plasma, serum and urine to be saved for later analysis of specific agents e.g. opiates, tricyclics
- **Lumbar puncture**
 - **Indications:**
 - Sepsis/bacterial meningitis; herpes simplex encephalitis, tuberculous meningitis and cause unknown.
 - Analyse the CSF for: MCS, Gram staining, Biochemistry, PCR...
 - **Contraindications:**
 - GCS 8 or less or deteriorating/ Focal neurological signs or abnormal posture
 - Prolonged seizure lasting 10 minutes or more and a GCS of 12 or less
 - Shock, Systemic meningococcal disease
 - Signs of raised ICP: unilateral or bilateral dilated pupils or sluggish pupillary reaction
 - Bradycardia or hypertension, Abnormal breathing pattern
- **Cerebral Imaging**
 - A CT scan is indicated if there is raised intracranial pressure, intracranial abscess or the cause of a decreased consciousness is unknown.
 - The CT scan may be normal, yet there may be raised intracranial pressure.
 - Clinical correlation is needed.

MANAGEMENT IN THE ED

- **General management is as per APLS guidelines.**
 - **Airway and Breathing:**
 - **Oxygen** should be administered.
 - **Consider intubation** in a child with a GCS of 8 or less or their GCS is deteriorating.
 - **Circulation**
 - Treat shock with a fluid bolus of **20 ml/kg** of crystalloid or colloid.
- **Disability**
 - GCS and pupillary examination,
 - Assess fontanelle, tone and posture.
 - Blood glucose is also needed.
- **Febrile seizures** occur from the age of 12 months to around 5 years of age. They should last less than 10 minutes and post-fit recovery should be relatively quick within 20 minutes, unless rescue medication has been given. Delayed fit recovery may indicate a more sinister pathology.
- **Non-convulsive status** can occur and should be considered if the child's GCS is not improving. Careful examination may reveal intermittent gaze deviation, nystagmus or other subtle signs to suggest a continued seizure. Use anticonvulsants as per protocol.
- **Exposure**
 - Front/back examination is required to look for:
 - A rash
 - Evidence of trauma
 - Drug use – check for powder residue

MANAGEMENT - SPECIFIC MANAGEMENT**1. Shock**

- Sepsis, trauma, anaphylaxis, hypovolaemia and heart failure may present with shock and reduced level of consciousness. For shock, fluid boluses are guided by clinical response, **up to 60 ml/kg** may be required. If more than 40 ml/kg is given, **inotropic or vasopressor** treatments should be considered, the child should be monitored on a critical care unit and intubation and ventilation considered. The appropriate specialists should be involved.

2. Sepsis

- Consider sepsis as a cause if:
 - Reduced consciousness
 - Abnormal vital signs, and
 - A temperature of greater than 38°C of ages <3 months, or
 - A temperature >39°C if 3-6 months, or
 - A temperature of less than 35.5°C

3. Trauma

- The child with a decreased consciousness and evidence of trauma should be managed as per APLS/ATLS and the NICE head injury guidelines. In the infant, trauma may not be immediately obvious, for example in the shaken baby. Children may be treated for other causes of reduced consciousness, e.g. sepsis, before the diagnosis is discovered.

4. Metabolic Illness

- Children may present in a variety of ways, including reduced level of consciousness.
- 4 main metabolic causes are briefly described here.
- **Hyperglycaemia**
 - **Diabetic ketoacidosis** is diagnosed if **capillary glucose is >11, pH<7.3 and ketones** in the urine. This should be managed as per the NICE guidelines.
- **Hypoglycaemia**
 - A metabolic cause should be considered if blood glucose **less than 2.6 mmol/l**.
 - Past medical history of consanguinous relationship should be sought.
 - **Treatment of hypoglycaemia**
 - As per APLS guidelines, the treatment is with **2 ml/kg of 10% dextrose bolus**.
 - An infusion of 10% dextrose solution should be given to maintain normal blood glucose: between 4-7 mmol/l.
- **Hyperammonaemia**
 - High ammonia levels are neurotoxic.
 - A plasma ammonia level of 200 micromol/l is significantly raised and needs actively treating.
 - The higher the peak or the longer the level remains high the worse the prognosis.
 - A level of 200micromol/l can be reduced with a **sodium benzoate infusion**.
 - If the levels remain high or are greater than 500 micromol/l, **emergency haemodialysis** is required.
- **Non-hyperglycaemic ketoacidosis**
 - Inherited conditions: organic acidopathies, amino acidopathies, fatty acid oxidative defects, mitochondrial electron transport chain defects and urea cycle enzyme defects can present as acute encephalopathy with a pH <7.3 and ketones in the urine. Further investigations include plasma lactate, plasma and urinary amino acids and urinary organic acids.
 - Advice should be sought from a metabolic medicine unit and careful monitoring of fluid balance to avoid raised intracranial pressure is required.

5. Intracranial Infection: Seven causes are outlined briefly here:

• BACTERIAL MENINGITIS

- Diagnosing meningitis may be difficult particularly in children <2 years.
- Classical symptoms such as decreased consciousness, neck pain/stiffness, vomiting and rash may be seen in the older child.
- Children, especially those <12 months old, may present as a fit with a fever.
- Clinical post-fit recovery may be delayed arousing suspicion of a non-benign cause.
- Treatment with **broad spectrum antibiotics** is needed until a definitive diagnosis can be made.
- Treating with **steroids** (dexamethasone 0.15 mg/kg prior to first dose of antibiotics) reduces profound hearing loss if the causative agent is Haemophilus influenza type B (HIB) and less so in cases of meningococcal or streptococcal infection.

• HERPES SIMPLEX ENCEPHALITIS

- Herpes simplex encephalitis should be suspected in a child with a decreased consciousness and focal neurological signs, fluctuating consciousness, contact with herpetic lesions or no obvious alternative cause.
- The diagnosis of herpes simplex encephalitis can be made with positive cerebrospinal fluid (CSF) for herpes simplex virus DNA in PCR of CSF.
- A positive PCR of CSF is highly sensitive and specific.
- A **MRI scan** and an **abnormal EEG** can strengthen the diagnosis. Nevertheless, waiting to perform a lumbar puncture should not delay treatment with intravenous acyclovir.

• INTRACRANIAL ABSCESS

- An intracranial abscess should be suspected in a child with headaches, altered behaviour, recent infection (ear, sinusitis etc.) or head trauma.
- Previous medical history of recent antibiotic use, neurosurgery or cyanotic congenital heart disease is relevant.
- Clinical signs may include a reduced consciousness, focal neurological signs, signs of sepsis and raised intracranial pressure. Diagnosis is with a **CT**.
- Treatment is with broad spectrum antibiotics and neurosurgical referral.

• TUBERCULOUS MENINGITIS

- Tuberculous meningitis should be suspected if the child has had contact with pulmonary tuberculosis.
- Diagnosis is with a positive **CSF PCR for TB DNA**.
- Further management should be guided by microbiology.

• RAISED INTRACRANIAL PRESSURE

- A child with decreased consciousness and raised intracranial pressure may have complaints of a headache, display altered behaviour and vomiting.
- Signs include unilateral or bilateral dilated pupils or unreactive pupils, abnormal posture, papilloedema, a relative bradycardia for age and hypertension.
- Manage the patient with the **head in the midline, raised 20 degrees** and aim to maintain normal physiology, which may require intubation and ventilation.
- Consider mannitol or hypertonic saline.

• HYPERTENSIVE ENCEPHALOPATHY

- Headache, nausea, vomiting, visual disturbances and focal neurological deficits and seizures may occur. Hypertension is defined as the systolic blood pressure >95th centile for age.
- Further management should be guided by a paediatric nephrologist or intensivist.
- Prolonged convulsion/post convulsive state
- Convulsions lasting more than 10 minutes need treating as per the APLS guidelines.
- In infants, in addition to the core investigations calcium and magnesium should be requested at presentation. Post convulsive state should last for less than one hour, if this is prolonged and the blood glucose is normal, the core investigations should be performed.

CHAPTER 7. PAEDIATRIC ABDOMINAL PAIN**Introduction**

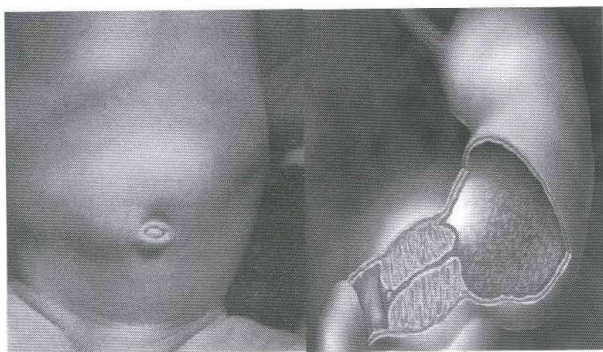
- Differential diagnosis of an acute abdomen based on age group

Age	Emergent	Non-Emergent
0-3 months	<ul style="list-style-type: none"> Necrotizing enterocolitis Volvulus Testicular torsion Incarcerated hernia Trauma Toxic megacolon Tumor 	<ul style="list-style-type: none"> Colic Acute gastroenteritis Constipation
3 month – 3 year old	<ul style="list-style-type: none"> Intussusceptions Testiculat torsion Trauma Volvulus Appendicitis Toxic megacolon Vaso-occlusive crisis 	<ul style="list-style-type: none"> Acute gastroenteritis Constipation Urinary tract infections HSP

I. HYPERTROPHIC PYLORIC STENOSIS

- Hypertrophic pyloric stenosis (HPS) causes a functional gastric outlet obstruction as a result of hypertrophy and hyperplasia of the muscular layers of the pylorus.
- In infants, HPS is the most common cause of gastric outlet obstruction and the most common surgical cause of vomiting.

Visible peristalsis
“Olive” shaped mass
 at RUQ



- THE CLINICAL FEATURES INCLUDE:**

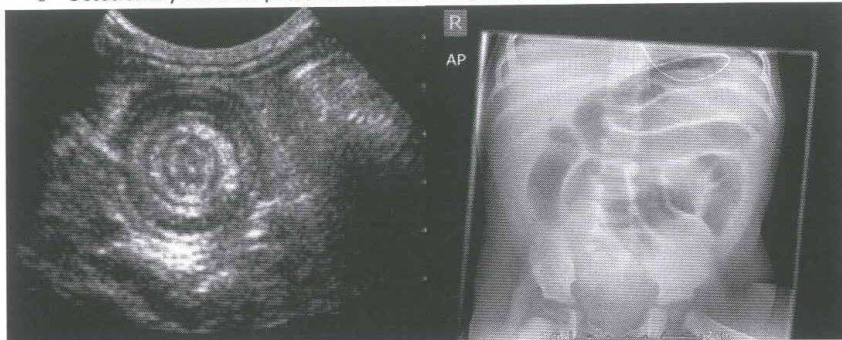
- Typical presentation is onset of initially **nonbloody**, always **nonbilious** vomiting at **4-8 weeks of age**.
- Although vomiting may initially be infrequent, over several days it becomes more predictable, occurring at nearly every feeding.
- Vomiting intensity also increases until pathognomonic **projectile vomiting** ensues.
- Slight hematemesis** of either bright-red flecks or a coffee-ground appearance is sometimes observed.

- o Patients are usually not ill-looking or febrile; the baby in the early stage of the disease **remains hungry and sucks vigorously** after episodes of vomiting.
- o Prolonged delay in diagnosis can lead to dehydration, poor weight gain, malnutrition, metabolic alterations, and lethargy.
- o Parents often report trying several different baby formulas because they (or their physicians) assume vomiting is due to intolerance.
- o An **olive-sized lump** may be palpable in the epigastrium (most prominent during a feed).
- **Diagnosis:**
 - o **Mainly clinical:** The child may develop **hypochloraemic alkalosis** and **hypokalaemia** due to recurrent vomiting of gastric contents.
 - o **Ultrasound:** Diagnostic of choice
 - o **Barium** upper GI study
 - o **Endoscopy**
- **ED MANAGEMENT**
 - o Directed at correcting the fluid deficiency and electrolyte imbalance
 - o Base fluid resuscitation on the infant's degree of dehydration
 - o Most infants can have their fluid status corrected within 24 hours; however, severely dehydrated children sometimes require several days for correction
 - o If necessary, administer an initial fluid bolus of **10 mL/kg with Hartmann's solution or 0.45 isotonic sodium chloride solution**
 - o Keeping the infant nil by mouth.
 - o Correction of electrolyte abnormalities, including hypoglycaemia.
 - o Referral to the paediatric surgical team for **pylorotomy**.

II. INTUSSUSCEPTION

- Intussusception is the invagination of one segment of bowel into an adjacent lower segment, causing bowel obstruction.
- With early diagnosis, appropriate fluid resuscitation, and therapy, the mortality rate from intussusception in children is less than 1%.
- If left untreated, however, this condition is uniformly fatal in 2-5 days.
- It typically affects children aged between **6 months and 4 years**.
- It may affect the small or large bowel, but most cases are **ileocolic**.
- Intussusception can rapidly compromise the blood supply to the bowel making relief of this form of obstruction urgent.
- Usually no underlying cause is found although there is some evidence that viral infection leads to enlargement of Peyer's patches, which may form the lead point of the intussusception. Occasionally a Meckel's diverticulum, polyp, or lymphoma is the lead point.
- **CLINICAL FEATURES INCLUDE:**
 - o **Vomiting:** Initially, vomiting is nonbilious and reflexive, but when the intestinal obstruction occurs, vomiting becomes bilious
 - o **Abdominal pain:** Pain in intussusception is colicky, severe, and intermittent
 - o **Passage of blood and mucus:** Parents report the passage of stools, by affected children, that look like "**Redcurrant jelly stool**"; this is a mixture of mucus, sloughed mucosa, and shed blood;

- o **Diarrhea** can also be an early sign of intussusception
- o **Lethargy**: This can be the sole presenting symptom of intussusception, which makes the condition's diagnosis challenging.
- o **Palpable abdominal mass** and occasionally a **'sausage-shaped'** mass may be visible.
- o **Dehydration and Pyrexia**.
- o Occasionally the child presents **shocked** without an obvious cause.

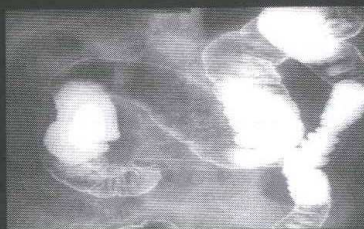


Target sign of intussusception on ultrasound

Distended bowel loops on PFA

Barium Studies

Show a classic "coiled spring" appearance due to trapping of contrast between layers of bowel.



• Diagnosis

- o **Radiography**: Plain abdominal radiography reveals signs that suggest intussusception in only 60% of cases
- o **Ultrasonography**: Hallmarks of ultrasonography include the **target sign** and **"pseudokidney signs"**.
- o **Barium enema**: This is the traditional and most reliable way to make the diagnosis of intussusception in children. A barium enema characteristically reveals a **'coiled spring' sign** or **sudden termination of the barium**. A barium enema is contraindicated if there is evidence of perforation, which requires surgical intervention.

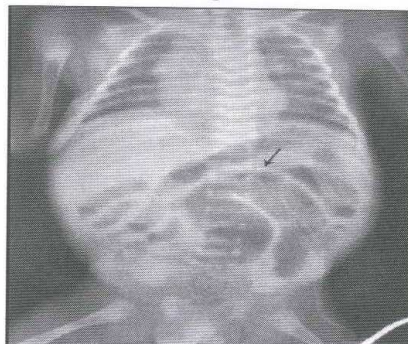
Management:

- o Refer to surgical team

Necrotizing Enterocolitis: Refer to Chapter 15/ Neonatal Emergencies.

III. VOLVULUS

- Volvulus may be due to congenital malrotations, Meckel's diverticulum, or adhesions from previous surgery.
- Congenital malrotations are the most frequent and result from the abnormal movement of the intestine around the superior mesenteric artery during embryological development.
- **Clinical features include:**
 - Abdominal pain.
 - Vomiting.
 - Abdominal distension.
- **Abdominal radiographs**
 - Will show a large, dilated loop of colon, often with a few air-fluid levels.
 - **Specific signs include:**
 - **Coffee bean sign**
 - **Frimann Dahl's sign** - three dense lines converge towards site of obstruction
 - **Absent rectal gas**



Coffee bean sign



Frimann Dahl's sign

IV. HIRSCHSPRUNG'S DISEASE

- Hirschsprung's disease is due to an absence of ganglion cells in a section of large bowel.
- It is usually confined to the rectosigmoid but may extend to involve the entire colon.
- The result is a section of bowel that is atonic.
- Examination of infants affected with Hirschsprung disease reveals **tyimpanitic abdominal distention** and symptoms of intestinal obstruction.
- Individuals in this age group may also present with **acute enterocolitis** or with **neonatal meconium plug syndrome**.
- Children with Hirschsprung disease are usually diagnosed by age **2 years**.
- Older infants and children with Hirschsprung disease usually present with **chronic constipation**.
- Upon abdominal examination, these children may demonstrate marked abdominal distention with palpable dilated loops of colon.
- Rectal examination commonly reveals an empty rectal vault and may result in the forceful expulsion of fecal material upon completion of examination.
- Children with suspected Hirschsprung's should be referred on to the paediatricians for further investigation. Ultimately the aganglionic section requires surgical excision.

CHAPTER 8. ACUTE LIFE-THREATENING EVENT**OVERVIEW**

- The term **Apparent Life-Threatening Event (ALTE)** applies to infants under 12 months with a history of a sudden event that is frightening to the observer and is characterised by some combination of: **Apnoea** (central or obstructive), **Colour change** (cyanotic, pallid or plethoric), **Change in muscle tone** (usually floppy but may be increased tone), **Choking or gagging**. These are frightening events for families and are diagnostic challenges for the physicians caring for these infants. It is frequently difficult to decide whether there has been a true life threatening event. Usually occurs in infants between **1 week and 10 months** and **most prior to 10 weeks**.
- ALTE occurs with highest frequency **in the first 3 months of life** but the term encompasses events up to 12 months of age. The term "**near-miss SIDS**" should be avoided as there is no proven association between ALTE and SIDS (Sudden Infant Death Syndrome).

CAUSES OF ALTE

- Despite the many causes of ALTE up to 50% of cases remain unexplained following a thorough evaluation.
- There are many causes of ALTE including:

<i>Less serious causes</i>	<i>More serious causes</i>
<ul style="list-style-type: none"> ▪ <i>Gastro-oesophageal reflux.</i> ▪ <i>Central apnoea</i> ▪ <i>Obstructive apnoea</i> ▪ <i>Breath-holding attack.</i> 	<ul style="list-style-type: none"> ▪ <i>Sepsis and/or meningitis.</i> ▪ <i>Lower respiratory tract infections.</i> ▪ <i>Seizures.</i> ▪ <i>Arrhythmias, e.g. long QT, SVT.</i> ▪ <i>Structural heart disease, e.g. duct-dependent lesions.</i> ▪ <i>Metabolic disorders.</i> ▪ <i>Non-accidental injury and Toxins.</i>

- **HIGH-RISK INFANTS**

- The following groups are identified as being at risk of having a significant underlying cause or a problematic course following an ALTE:
 - *Age < 28 days*
 - *Significant prematurity*
 - *Significant prior medical illness*
 - *Clinically unwell looking*
 - *Recurrent events before presentation*
 - *More severe/prolonged ALTE symptoms*

ASSESSMENT AND INITIAL INVESTIGATIONS FOR ALTE

- **HISTORY**

- History should ideally be obtained from the person(s) who observed the infant during or immediately after the event.
- **Description of event:**
 - What attracted the caregiver's attention?
 - Activity at the time of the event (awake/asleep)
 - Colour (cyanosis, pallor, plethora) and colour distribution (whole body vs. perioral)
 - Tone: floppy, stiff, normal
 - Abnormal movements including eye movements
 - Time and duration of event
 - Blood/fluid in nose or mouth

- **Circumstances and environment prior to event:**
 - Relationship of event to feeding or vomiting
 - Sleep position: supine, prone, side
 - Environment: nature of sleeping arrangement (cot, car seat, bed etc.), type of bedding, type of clothing
- **Recent illness and other important points in the history:**
 - History of coryza or other URTI/LRTI symptoms in infant or family
 - **Relevant PMH:** prematurity, vaccinations, significant previous illnesses
 - Family history of Sudden Unexpected Death in Infancy or later deaths, parental consanguinity
 - **Social factors:** family known to Social Services or on Child Protection Plan, parental smoking, drug and alcohol use, previous ED attendances
- **Interventions used by caregiver:**
 - Degree of resuscitation required: gentle or vigorous stimulation, mouth-to-mouth, chest compressions (layperson or medically trained)

● EXAMINATION

- A head-to-toe examination should be performed including plotting growth and head circumference. Consider using a body map if there is a suspicion of child abuse.

INVESTIGATIONS

- Directed by the findings of a thorough history and examination and may include:
 - **ECG:** for evidence of long QT or pre-excitation.
 - **Blood glucose.**
 - **ABG/VBG:** to assess for acidosis and lactate.
 - **FBC:** looking for evidence of a systemic infection.
 - **U&E:** looking for evidence of hyponatraemia or hyperkalaemia.
 - **Cultures:** as directed by the clinical picture e.g. urine, stool, blood, etc.

ED MANAGEMENT OF ALTE

- This may include initial resuscitation and/or management of any underlying aetiology for the presentation (e.g. infection, NAI etc.)
- *Most children will require admission for close monitoring with apnoea and pulse oximetry monitoring for up to 24 hours.*
- Discharge of a child following an ALTE may be considered if:
 - *The episode is single, short and self-limiting*
 - *It was related to feeding*
 - *A normal feed has been observed*
 - *Advice about appropriate feed volumes has been given*
 - *There is no abnormality detected on examination*
 - *Parental anxiety has been addressed*
- These patients should have early follow-up with their GPs and in practice most babies are admitted for observation.
- Parents should be taught **basic paediatric life support** prior to discharge.
- Note there is no evidence that apnoea monitors save lives.
- Although ALTE is not predictive of Sudden Infant Death Syndrome (SIDS), the opportunity should be taken to educate parents about practices that have been shown to lower the incidence of SIDS, namely: *avoiding exposure to tobacco smoke, safe infant sleeping practices (on their back, face uncovered, firm mattress, no loose bedding or toys), avoiding bed-sharing.*

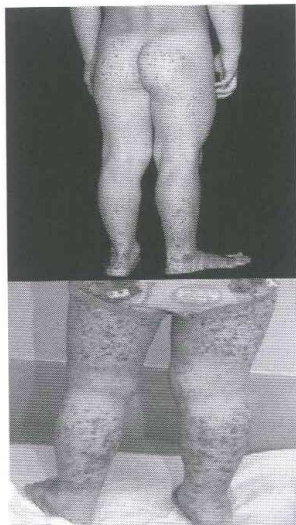
CHAPTER 9. BLOOD DISORDERS

• CAUSES OF PURPURA INCLUDE

- Meningococcal disease
- Henoch–Schönlein purpura
- Thrombocytopenia, e.g. ITP
- Leukaemia, Septic shock, or Aplastic anaemia
- Enteroviral infection/ Trauma
- Forceful coughing or vomiting.

I. HENOCH-SCHÖNLEIN PURPURA (HSP)

- **Henoch–Schönlein purpura** (also called **IgA vasculitis (IgAV)**) is a vasculitic condition that affects the small arteries of the kidneys, skin, and gastrointestinal tract.
- It usually affects children between the **ages of 3 and 10 years old**.
- It is twice as common in boys, peaks during the winter months, and is often preceded by an upper respiratory tract infection.
- **CLINICAL FEATURES INCLUDE:**
 - **Rash:** erythematous macules develop into purpuric lesions, which are characteristically concentrated over the buttocks and extensor surfaces of the lower limbs.
 - **Arthralgia:** particularly the knees and ankles.
 - **Peri-articular oedema**
 - **GIT:** Abdominal pain, N&V, Bloody diarrhoea
 - **Haematuria:** due to glomerulonephritis



ED INVESTIGATIONS

- **Urinalysis:** which may reveal micro- or macroscopic haematuria, and/or proteinuria.
- **U&E:** occasionally nephrotic syndrome and renal failure develop.
- **Blood pressure:** hypertension is a risk factor for progressive renal disease.
- **FBC:** to ensure a normal platelet count and exclude thrombocytopenia as a cause.

COMPLICATIONS

- **Renal failure:** involvement occurs in 50% of older children but is only serious in approximately 10% of patients. Less than 1% of patients with HSP progress to end-stage kidney disease.
- **Intussusception** (in 2-3% of patients),
- **Gastrointestinal bleeding,**
- **Bowel infarction,**
- **Myocardial infarction,**
- **Pulmonary haemorrhage,**
- **Pleural effusion,**
- **Seizures**
- **Mononeuropathies.**

ED MANAGEMENT OF HSP

- Mainly symptomatic:
 - Adequate oral hydration/bed rest
 - Symptomatic relief of joint and abdominal pain:
 - **Naproxen 10 to 20 mg/kg BD** (maximum 1500 mg per day)
 - **Oral Prednisone 1 to 2 mg/kg per day** (maximum dose of 60 to 80 mg per day)
 - **IV Methylprednisolone 0.8 to 1.6 mg/kg per day**(maximum dose of 64 mg/day)
 - Paediatric referral.
- **Hospitalization is indicated in:**
 - Fail to maintain oral hydration and require the administration of intravenous fluids.
 - Patients who have significant gastrointestinal bleeding, severe abdominal pain, changes in mental status, severe joint involvement limiting ambulation and/or self-care, or evidence of significant renal disease (elevated creatinine, hypertension, or proteinuria).

II. SICKLE CELL DISEASE IN CHILDREN

OVERVIEW

- Sickle cell disease is caused by **HbS hemoglobinopathy** which produces rigid, distorted and dysfunctional erythrocytes called sickle cells.

CAUSE

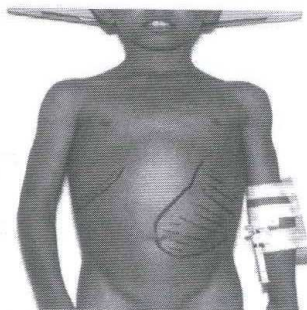
- **Types of sickle cell disease**
 - Sickle cell anemia (homozygous SS genotype)
 - Sickle beta thalassemia
 - Sickle HbC disease

PRECIPITANTS

- **Commonly:**
 - Infection/ Dehydration/ Hypoxia
 - Drugs (e.g. Sedatives, local anaesthetics)
 - Functional asplenia typically develops in childhood. Prophylactic treatment with hydroxyurea can cause neutropenia and cardiomyopathy.
- Patients with sickle cell disease are at risk of infection due to underlying immunosuppression.

PRESENTATIONS

- Types of sickle cell crisis presentations:
 - *Infections*
 - *Vaso-occlusive crisis*
 - *Acute chest syndrome*
 - *Acute splenic sequestration*
 - *Aplastic crisis*
 - *Stroke*
 - *Priapism*
 - *Bone pain: The long bones of the extremities are often involved, often due to bone marrow infarction*
 - *Anemia: Universally present, chronic, and hemolytic in nature*
 - *Hand-foot syndrome: This is a **dactylitis** presenting as bilateral painful and swollen hands and/or feet in children*



Enlarged spleen in a child with sickle cell anemia.

- *Ophthalmologic involvement: Ptosis, retinal vascular changes, proliferative retinitis*
- *Cardiac involvement: Dilatation of both ventricles and the left atrium*
- *GI involvement: Cholelithiasis is common in children; liver may become involved*
- *GU involvement: Kidneys lose concentrating capacity; priapism is a well-recognized complication of SCD*
- *Dermatologic involvement: Leg ulcers are a chronic painful problem*
- Pain crises begin suddenly. The crisis may last several hours to several days and terminate as abruptly as it began. The pain can affect any body part. It often involves the abdomen, bones, joints, and soft tissue, and it may present as **dactylitis** (bilateral painful and swollen hands and/or feet in children), acute joint necrosis or avascular necrosis, or acute abdomen. With repeated episodes in the spleen, **infarctions and autosplenectomy** predisposing to life-threatening infection are usual. The liver also may infarct and progress to failure with time. Papillary necrosis is a common renal manifestation of vaso-occlusion, leading to isosthenuria (ie, inability to concentrate urine).

ASSESSMENT

- Always consider the presence of all types of sickle cell crisis, regardless of the dominant presentation:
 - Symptoms and signs of local and systemic infection
 - Respiratory signs and symptoms/Increasing spleen size
 - Shock and evidence of organ failure/ Baseline and current Hb
 - CXR if fever, chest pain or hypoxia/ CT Head if stroke suspected
 - Consider MSU, FBC, reticulocytes, bilirubin, hemolytic screen and cross-match
 - Abdominal ultrasound if crisis is in abdomen.
 - Blood film: sickle cells and evidence of hemolysis (e.g. target cells, schistocytes)

GENERAL MANAGEMENT OF SICKLE CELL DISEASE IN THE ED

- **Immediate treatment in the ED**
 - **Assess pain and give analgesia:**
 - **Morphine;** Assess 20 minutes after administration.
 - If more than one dose is required, use a **Diamorphine 10 mcg/kg/hour** infusion pump. Start with a high dose and reduce once pain control is achieved.
 - **Keep patient warm and well hydrated,** Reassure patient:
 - **IV fluid: Dextrose-saline with KCL.**
 - **Assess O₂ saturation:** Use O₂ via face mask if necessary.
 - **Antibiotherapy:**
 - Is patient taking Penicillin regularly? If not, consider **IV Penicillin**.
 - Advise to **double dose of Penicillin** at sign of infection or start of crisis.
 - If already taking Penicillin change antibiotic to **Cephalosporin**.
 - If Penicillin and Cephalosporin not acceptable, then give **IV Erythromycin** - give slowly as it can be irritant to veins.
 - **Anti emetic:** Metoclopramide.
 - **Reassess pain:** If pain settles after 1 hour, discharge home.
 - Discuss all with haematology (for follow up).
 - Admit if:
 - *The pain is not controlled after 1 hour, or is severe*
 - *Patient is pyrexial or signs of infection. (Most sickle cell crises are precipitated by infection.)*

SPECIFIC TREATMENT

A. ACUTE CHEST SYNDROME

- Acute chest syndrome is sequestration within the lungs. It is characterised by:
 - *Pyrexia (The temperature > 38°C)*
 - *Chest pain or acute respiratory distress*
 - *Often bilateral lung consolidation*
 - *Tachycardia and tachypnoea.*
 - *Cough is a late symptom*
- Hypoxia sets up a vicious cycle of sickling within the lungs.
- Very difficult to reverse, early prompt and effective treatment is vital.
- This is a haematological emergency and must be discussed with haematology.
- **MANAGEMENT OF ACUTE CHEST SYNDROME**
 - Oxygen - monitor with pulse oximeter
 - Blood gases/ Urgent CXR
 - IV antibiotics - Penicillin and Cephalosporin
 - IV fluids & Analgesia
 - Inform Haematologist on duty as exchange transfusion may be indicated
 - Inform Consultant Anaesthetist as ventilation may be necessary.

B. APLASTIC CRISIS

- The production of red cells by the bone marrow may be reduced after an infection.
- An individual with sickle cell relies on the constant activity of the bone marrow to produce enough red cells to survive.
- The life span of their blood cells is 15 - 20 days, if there is a rapid fall in the Hb without reticulocyte response, this should be taken very seriously.
- Therefore, an aplastic crisis can be lethal.
- **Parvovirus** which presents like influenza is the usual cause of an aplastic crisis that follows an infection.
- *Check: Hb, Reticulocyte count, Folate level, Parvovirus antigen & Antibody titre.*
- *Transfuse if no reticulocyte response, but inform Haematologist first.*

C. ACUTE SPLENIC SEQUESTRATION

- Acute splenic sequestration is caused by the spleen enlarging during the crisis and results from massive sickling in the spleen and hepatic sinuses.
- There is a precipitate fall in the patient's normal haemoglobin level of **more than 2 g/dl** from steady state and a marked increase of reticulocytes in the peripheral blood.
- Acute splenic sequestration is characterised by:
 - *Sudden onset of tachypnoea,*
 - *Pallor,*
 - *Abdominal pain and*
 - *Splenic enlargement.*
- This may be precipitated **by fever, dehydration and hypoxia.**
- Rapid sequestration of the red cells leads to **sudden anaemia** and **death** from hypoxic cardiac failure with pulmonary oedema.
- This is most common in children and infants under the age of 5 years.
- It is useful to teach the parent(s) to palpate the spleen in these children so that if they become ill and the spleen enlarges they know that they must get the child to hospital quickly.

- **Investigations:**

- Hb, Reticulocytes, Group and X-Match, Blood Cultures, WBC, U&E's.

- **MANAGEMENT OF ACUTE SPLENIC SEQUESTRATION:**

- **IV access**
- **Packed red cells transfusion** without delay
- If shocked, **start colloid infusion** while waiting for blood
- **Broad spectrum antibiotics** - *IV Penicillin and Cephalosporin*. This should offer some protection against pneumococcus and haemophilus influenza
- If breathless - **urgent CXR**
- Inform Haematologist on-call.

D. PRIAPISM IN SICKLE CELL DISEASE

- This is a painful persistent erection of the penis caused by intravascular sickling in the erectile tissue.
- If priapism persists for more than 12 hours this can lead to damage of the erectile tissue which results in the patient being unable to get an erection. This can lead to **permanent impotence**.

- **MANAGEMENT OF PRIAPISM IN SICKLE CELL SYNDROME**

- Give **adequate analgesia**
- **Reassure** and keep the patient **warm**
- Re-hydrate - **IV fluids**
- Contact Urologist and Haematologist immediately
- Sedate? **IV Diazepam**
- **Group and save** - consult Haematologist before transfusing
- Surgical intervention may be necessary if there is no improvement
- **Do not use ice packs**

E. OSTEOMYELITIS

- Consider particularly if any localised pain fails to resolve within 48 hours. Osteomyelitis most often results from infection with **Haemophilus, staphylococcus or salmonella**.
- If no resolution of fever or swelling **within 48 hours**:
 - *Repeat Blood cultures/ X-Ray affected area*
 - *Review antibiotics/ Obtain an orthopaedic opinion.*
- **Admission to ward from the ED**
 - Patients usually go to the haematology team
 - Inform Haematologist as soon as possible

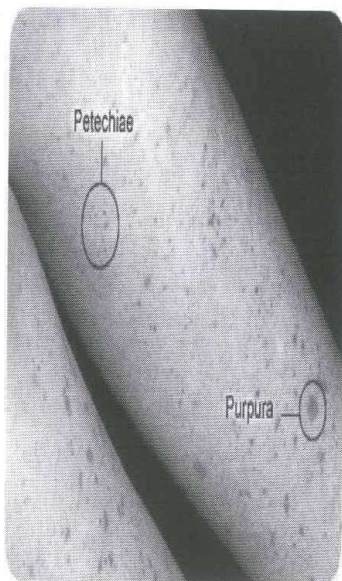
Hand Foot Syndrome: Dactylitis

- Early complication of SCD
- Highest incidence 6 months to 2 years
- Painful swelling of hands and feet
- Treatment involves fluids and pain medication
- Fevers treated as medical emergency



III. IMMUNE THROMBOCYTOPENIC PURPURA

- Idiopathic (Immune) thrombocytopenic purpura (ITP) is a clinical syndrome in which a decreased number of circulating platelets (**thrombocytopenia**) manifests as a **bleeding tendency, easy bruising (purpura)**, or **extravasation of blood** from capillaries into skin and mucous membranes (**petechiae**).
- In many cases ITP's cause is **autoimmune** not idiopathic, with antibodies against platelets being detected in approximately 60% patients. A preceding illness is the usual likely trigger.
- **Clinical features include:**
 - *Purpuric rash.*
 - *Mucous membrane bleeding*
 - *Conjunctival haemorrhage*
 - *Occasionally gastrointestinal bleeding*
- *Children may be suspected of suffering from non-accidental injury due to the ease of bruising and bleeding*
- **Note—the presence of lymphadenopathy, hepatomegaly or splenomegaly suggests an alternative diagnosis e.g. leukaemia.**



- The most important investigation is a **full blood count**. Platelets will be $< 30 \times 10^9/L$.
- Children should be referred on for paediatric review.
- Treatment is usually expectant because most cases resolve spontaneously over 3 months.
- Occasionally life-threatening haemorrhage occurs and patients should be managed in the usual ABCDE manner and resuscitated accordingly.

CHAPTER 10. CONCERNING PRESENTATIONS

I. APPROACH TO NON-ACCIDENTAL INJURIES

- Non-accidental injuries in the pediatric population can be easily missed unless health care professionals are attuned to subtle clues in both the history and physical exam.
- *Non-accidental injuries should be suspected when an **injury is unexplained**, the severity of the injury is **incompatible with the history**, the **history keeps changing**, or the injury is **inconsistent with the developmental age of the child**.*
- **Any delay is seeking medical care** following an injury is also suspicious for abuse.
- Suspected abuse needs to be meticulously investigated with the assistance of child protection services.

RISK FACTORS FOR CHILD ABUSE

- The following factors may increase the likelihood of abuse:
 - **Special needs children:** Developmental Delay, Cerebral Palsy, Prematurity
 - **Social disadvantage:** *Low income, Inadequate housing, being discriminated against because of ethnicity, religion, disability, or sexual orientation.*
 - **Stress:** *possibly caused by **financial problems** and difficulties in the **parents' relationships**. This can reduce some adults' ability to control aggressive feelings towards their children or to care for their children properly.*
 - **Single Parent**
 - **Substance abuse**
 - **Mental illness/ Domestic violence/ Former victims of abuse**

FEATURES OF POSSIBLE CHILD ABUSE

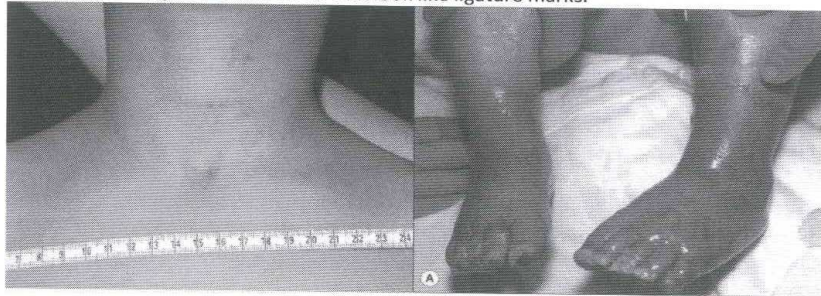
- *Delay in seeking medical attention with no adequate explanation.*
- *Injuries inconsistent with the history given.*
- *Injuries inappropriate for the developmental age.*
- *Vague history or lacking details.*
- *Changing history—either between parents/carers/child or alterations over time.*
- *Abnormal parental attitudes (e.g. lack of parental concern).*
- *Frequent ED attendances—Children with three or more attendances for different conditions in the past year should be referred on to the Community Health Visitor Team or Social Worker Team **within 5 days** of attendance (RCEM standard).*

NICE CLINICAL GUIDELINE 89

1. BRUISING & LACERATIONS

- The most common manifestation of abuse is **bruising**.
- Suspect child maltreatment if a child or young person **has bruising in the shape** of a hand, ligature, stick, teeth mark, grip or implement.
- Suspect child maltreatment if there is bruising or petechiae (tiny red or purple spots) that are **not caused by a medical condition** (for example, a causative coagulation disorder) and if the explanation for the bruising is unsuitable.
 - Bruising in a child who is not independently mobile
 - Multiple bruises or bruises in clusters
 - Bruises of a similar shape and size
 - Bruises on any non-bony part of the body or face including the eyes, ears and buttocks
 - Bruises on the neck that look like attempted strangulation
 - Bruises on the ankles and wrists that look like ligature marks

- Suspect child maltreatment if there is a report or appearance of a **human bite mark** that is thought unlikely to have been caused by a young child.
- Consider neglect if there is a report or appearance of an **animal bite** on a child who has been inadequately supervised.
- Suspect child maltreatment if a child has lacerations, abrasions or scars and the explanation is unsuitable. Lacerations, abrasions or scars:
 - On a child who is not independently mobile
 - That are multiple with a symmetrical distribution
 - On areas usually protected by clothing (for example, back, chest, abdomen, axilla, genital area)
 - On the eyes, ears and sides of face
 - On the neck, ankles and wrists that look like ligature marks.



2. BURNS

- An estimated 15-25 % of pediatric burns are the result of abuse.
- Like bruising, the burn injury pattern and location can be a red flag for abuse.
- Children tend to explore objects with the palmer surface of their hand and will not touch a heated object very long.
- Suspect child maltreatment if a child has burn or scald injuries:
 - On any soft tissue area that would not be expected to come into contact with a hot object in an accident (for example, the backs of hands, soles of feet, buttocks, back) or
 - In the shape of an implement (for example, cigarette, iron) or
 - That indicate forced immersion, for example:
 - Scalds to buttocks, perineum and lower limbs
 - Scalds to limbs in a glove or stocking distribution
 - Scalds to limbs with symmetrical distribution
 - Scalds with sharply delineated borders.

3. FRACTURES

- Fractures are the second most common manifestation of child abuse after soft tissue injuries.
- 85% of fractures from non-accidental injury occur in children **under the age of 3** and 69% occur in infants **under the age of 1**.
- Although there are fracture patterns suggestive of abuse, **there are no patterns that are pathognomonic for non-accidental injury**.
- *Any fracture in a young child should be concerning, especially if the child is not ambulating.*

- If an infant is **pulled or wrenched**, the corner of the metaphysis can be torn, commonly referred to as a **"bucket handle" fracture**.
 1. **Metaphyseal corner fractures**, also known as **classical metaphyseal lesions (CML)** or **bucket handle fractures**, are observed in young children, less than 2 years old. It is considered pathognomic for **non-accidental injury (NAI)**.
 2. **Finding spiral fractures** in the bone shaft is indicative of a twisting injury rather than a transverse fracture from direct impact.
 3. **Femur fractures prior to the age of walking** are especially concerning, as are bilateral long-bone fractures.
 4. Violent squeezing of an infant's chest results in **anterior and/or posterior rib fractures** which are difficult to acquire with other injuries as children's ribs are flexible.
 5. Additionally, **fractures of the sternum, scapula, or spinous processes** are unusual in the pediatric population.
 6. **Skull fractures** result from a direct force on the calvarium and are very uncommon in children less than 18 months. In non-accidental trauma, they most commonly occur as linear fractures in the parietal bone and can often be complex in nature.



Bucket handle fractures of NAI

Spiral fracture of NAI

- A significant red flag for NAI is finding multiple fractures at various stages of healing..

4. ABUSIVE HEAD TRAUMA / SHAKEN BABY SYNDROME

- Abusive head trauma, also known as **shaken baby syndrome**, is the most common cause of child abuse death, usually occurring during the first year of life.

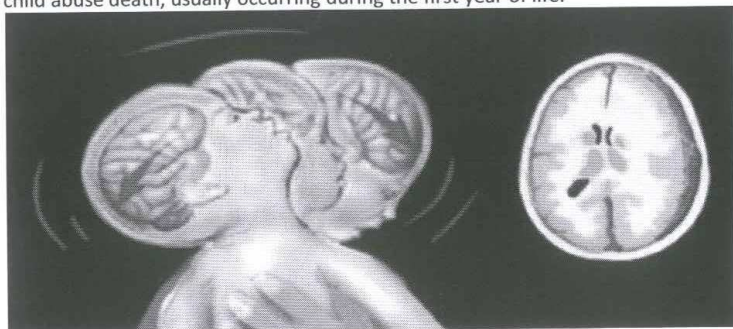


Figure 5.10.1. Shaken Baby Syndrome

- Shaking or blunt head trauma can result in cranial injuries such as **unilateral or bilateral subdural hemorrhage, diffuse retinal hemorrhage, and diffuse brain injury.**
- **Retinal hemorrhages**, often multilayered, occur in 60-85 % of non-accidental head injuries and are uncommon in accidental head trauma.
- The diagnosis of abusive head trauma is often missed since often no history of head trauma is provided and the signs and symptoms may be non-specific, such as vomiting, poor feeding, irritability or lethargy.
- Some caregivers may only present for medical care if more severe symptoms arise such as seizure, apnea or coma. The majority of children have an abnormal neurological exam but many will have no external signs of injury.

5. SEXUAL ABUSE

- Sexual abuse is when a child or young person, male or female, is pressurized, forced, or tricked into taking part in any kind of sexual activity with an adult or young person.
- This can include **kissing, touching the young person's genitals or breasts, intercourse, or oral sex.** Encouraging a child to look at **pornographic magazines, videos or sexual acts** is also sexual abuse.
- In the UK, 1% of children experience sexual abuse by a parent or carer, 3% by another relative, 11% by people known but unrelated to them, and 5% by an adult stranger or someone they have just met.
- Most children who present with an allegation or a history of sexual abuse do not have any physical signs when examined. This is because the abuse may have taken place some time before the examination and injuries to the genital area heal very quickly, or because the abuse was non-penetrative (e.g. kissing or touching).
- **Physical signs that may be present include:**
 - *Injury to the genitalia or anus.*
 - *Perineal pain, discharge, or bleeding.*
 - *Pregnancy.*
 - *Sexually transmitted infections.*
- The child's behaviour may suggest sexual abuse if he or she:
 - Becomes anxious about going to a particular place or seeing a particular person.
 - Suddenly starts having behaviour problems, such as being aggressive.
 - Suddenly starts having extreme mood swings, such as brooding, crying, or fearfulness.
 - Has a sudden deterioration in school results.
 - Displays unexpectedly explicit sexual knowledge for their age.
 - Starts wetting the bed again, having previously been dry by night.
 - Engages in risky behaviours—drug-taking, unprotected sex with numerous partners (in an adolescent).
 - Starts harming him or herself.

6. CHILD NEGLECT

- About 6% of children in the UK experience a serious absence of care at home during childhood. Neglect is the persistent lack of appropriate care of a child.
- This includes lack of love, stimulation, safety, nourishment, warmth, education, and medical attention. Neglect may be suspected if the mechanism of injury suggests lack of appropriate supervision.
- Child neglect is not always deliberate.

- Sometimes, a caregiver becomes physically or mentally unable to care for a child, such as in untreated depression or anxiety.
- Other times, alcohol or drug abuse may seriously impair judgment and the ability to keep a child safe.
- **Physical signs of neglect include:**
 - *Ill-fitting, dirty clothes and shoes, not dressed warmly enough in cold weather.*
 - *Appearing very dirty, with matted and unwashed hair or smelling bad*
 - *Untreated or delayed treatment for illnesses and physical injuries.*
 - *Failure to thrive, gain weight, and meet developmental milestones.*

7. EMOTIONAL ABUSE

- Parents from all types of backgrounds may emotionally abuse their children. About 6% of children in the UK experience frequent and severe emotional maltreatment during childhood. Emotional abuse is the hardest type of abuse to define precisely but occurs when a parent or carer behaves in a way that is likely to seriously affect their child's emotional development.
- Physical abuse, sexual abuse, and neglect nearly always involve an element of emotional abuse. However, emotional abuse can be the only form of abuse a child is suffering.
- It can range from constant rejection and denial of affection through to continual severe criticism. It is not a one-off outburst but continues over time.
- It's not always easy to identify when a child is being emotionally abused. Some of the ways children react to emotional abuse are:
 - *Having low self-confidence and a poor self-image*
 - *Being withdrawn, unable to trust others, and having difficulty forming relationships.*
 - *Being delayed emotionally, socially, or academically.*
 - *Becoming anxious, depressed, demanding, aggressive, destructive, or even cruel.*

CLINICAL ASSESSMENT

• HISTORY

- To begin the history, open ended questions should be used to determine how the injury occurred. These questions should be directed first to the child, if they are able to answer, and then to the parent.
- During this portion of the interview it is very important to assess the parent-child interaction. If there is a suspicion of non-accidental injury, it is important to then interview the child alone and if the caregiver will not permit this it is considered a red flag for abuse. Concerning information during history taking is if the injury is not well explained, the severity does not match with the history, or the injury is inconsistent with the developmental age (**e.g. a 1 month old baby rolled off the couch**).
- If there are inconsistencies with the history or any delays in seeking medical care, these are also of concern.

• PAST MEDICAL HISTORY

- In addition to the history of current events, a past medical history for the child should be established.
- For example, was the child premature, do they have any significant medical or developmental problems? This helps determine if the child has a possible medical cause for the presentation (e.g. osteogenesis imperfecta) or if they have risk factors for abuse (e.g. prematurity, physical or learning disabilities).

• SOCIAL HISTORY

- Establish the family structure: which adults are at home? Are there any siblings and what are their names and ages? Does the child have a minder or attend nursery or playgroup? Which nursery/school does the child attend?
- Are they registered with a GP? Have the family had contact with social services before?
- Is there a history of domestic violence? Is there a history of illness in the parents/carers (e.g. postnatal depression, alcohol abuse)?

• PHYSICAL EXAM

- A general and focused physical exam needs to be completed.
- Findings that raise suspicion are injuries inconsistent with the history, multiple injuries in various stages of healing, or any injuries pathognomonic for abuse, **such as cigarette burns, bucket handle fracture...** Evidence of poor caretaking, sudden onset of mental status changes, any bruises on an infant that is not cruising yet, bruises to the pinna, neck or abdomen and any injury to the genitalia.
- It is important to keep in mind that some findings may be concerning initially but are actually not signs of intentional injury:
 - **Intense crying, coughing or retching may cause petechiae on the face and shoulders,**
 - **Mongolian spots may appear as bruising in the lumbosacral area,**
 - **Coagulopathies may result in usual bruising, and**
 - **Pathologic bone disease (e.g. chronic renal disease, osteogenesis imperfecta, and rickets) may present with suspicious fractures.**

INVESTIGATIONS

• Laboratory investigations

- Need to be tailored to the nature of the injury and findings on physical exam.
- In all cases of bruising, **screening tests for a bleeding diathesis** should be performed with **FBC, INR, and PTT.**
- A more extensive evaluation for a bleeding disorder may include **fibrinogen, von Willebrand's testing, factor deficiencies and homocysteine levels.**

• Both a skeletal survey, which consists of a series of radiographs of the skull, thorax, long bones, hands, feet, pelvis, and spine, and a radionucleotide bone scan are tools to reveal sites of past injury.

• INDICATIONS FOR A SKELETAL SURVEY ARE:

- *Any child less than 2 years of age with any evidence of abuse,*
- *Any child less than 5 years of age with a suspicious fracture, or*
- *Any older child who is unable to communicate areas of pain or trauma (e.g. intellectually disabled).*
- In the event that a skeletal survey is negative but the suspicion remains high, a radionucleotide bone scan can be performed.
- **A skeletal survey may also be repeat 7 – 10 days** following the injury to reveal healing fractures that may have been missed.
- This is a significant amount of radiation for a child to be exposed to and thus the decision to complete a skeletal survey needs to be made carefully.
- If a head injury is suspected a **head CT and ophthalmologic exam** are important.
- If there are any concerns about abdominal trauma, **liver and pancreatic enzymes** should be ordered along with an **abdominal CT** and both **urine and stool** needs to be screened for blood.

II. LEGAL FRAMEWORK FOR CHILD PROTECTION

- Everyone who deals with children is responsible for safeguarding and has a legal obligation to raise any concerns they may have about a child's welfare.
- The Children Act 1989 contains most of the relevant law relating to child protection.
- **Section 47 of the Children Act 1989** covers children at risk or suffering harm, from physical, sexual, or emotional abuse, or neglect.
- If there is significant concern that a child is at risk of harm, then social services will instigate **Section 47** and contact the police.
- Social services, the police, and the paediatricians will then formulate a joint strategy plan to decide if the child needs to be taken into care or can be discharged home.
- If it is felt that a child needs to be in a place of safety, the parents can voluntarily allow the child to be taken into care, which may be the hospital or a close relative (**Section 20 of the Children Act**).
- If the parents insist on taking the child out of hospital when there is concern for the child's welfare, the police should be informed.
- **Under Section 46 of the Children Act**, the police have the power to enforce a police protection order that can keep the child in a designated place of safety for **up to 72 hours**.
- The social worker can apply for an extension to this in the form of an emergency protection order (**Section 44 of the Children Act**).
- The Children's Act 2004 requires each local authority in England and Wales to promote cooperation between different agencies involved in the welfare of children.
- It also requires them to establish Local Safeguarding Children Boards of which NHS Trusts are statutory members. Every NHS Trust will have a named doctor for Child Protection who is responsible for promoting and advising on safeguarding issues.

ED MANAGEMENT OF A CHILD WITH A CONCERNING PRESENTATION

- The immediate management should involve **ensuring the child is pain-free and treating any injuries or illness appropriately**.
- **Meticulous documentation is essential**. Notes should be factual (e.g. 4 × 1cm round bruises found on the medial aspect of the left upper arm) and not attribute blame or causation (e.g. finger imprints found on medial aspect of left upper arm).
- Documenting injuries in a diagram is a useful way to capture information.
- If sexual assault is suspected, a **genital examination should not be pursued in the ED**. This should be performed only once, by a senior clinician in child protection, in collaboration with a police surgeon (clinical forensic physician).
- Further information should be gathered about the child. For example: checking whether the child or any siblings are known to social services or whether they are subject of a Child Protection Plan; looking up previous ED attendances; contacting the GP to gain a past medical history for the child and background information on the family (e.g. parental mental health or substance misuse issues). Any suspicion of abuse should prompt early involvement of an expert senior doctor, e.g. ED consultant and/or paediatrician.
- Once information has been gathered and the case has been discussed/reviewed with a senior clinician the level of concern can be established e.g. **no concern; minor concern or unsure; more than a minor concern**.
- The level of concern determines the ongoing management:
 - **No concern:** a routine notification **letter of the child's ED attendance should be sent to the GP**. Plus, a letter of notification should be sent **to the midwife if the child is <10**

days old (faxed urgently); the **Health Visiting Team**, if the child is aged 10 days—5 years (pre-school children); or the **School Nurse** for school children (within 5 days). This is the standard recommended by **Lord Laming's report in 2009** (The Protection of Children in England: a progress report), the Government's report Working Together to safeguard children 2006, and the College of Emergency Medicine Best Practice Statement for Safeguarding Children.

- **Minor concern or unsure**: should have a **senior Emergency Medicine opinion** and then be referred to the **ED Liaison Health Visitor the next working day**.
- **More than a minor concern**: should be referred **directly for a senior paediatric opinion** and **referred to social services** (the local Trust Child Protection Policy should be followed for this).

III. AGENCIES IN SAFEGUARDING CHILDREN

1. CHILDREN'S SOCIAL CARE (CSC)

- Commonly referred to as social services, take the lead in investigating and managing child protection cases.
- They will know whether or not a child and/ or family have been previously involved with children's social care.

2. POLICE

- Safeguarding children is a fundamental part of the duties of all police officers.
- All forces have child abuse investigation units who undertake criminal investigations in cases of suspected child abuse. The child abuse investigation team will have knowledge of any previous criminal involvement of child/parents/carers.

3. EDUCATION

- Schools have a statutory responsibility, like healthcare organisations, to safeguard children and young people. **School teachers** will have a good knowledge of a child's day to day demeanour and developmental/academic strengths and weaknesses.
- **The school nurse** will be aware of issues relating to health and development as well as other issues, if any, affecting the parents or other children in the family.

4. HEALTH

- All layers and elements of the health service have a statutory responsibility to safeguard children and young people.
- This includes **health visitors, GPs, staff in secondary and tertiary healthcare**, e.g. specialist hospitals, private hospitals, mental health services, genitourinary and family planning services, dentists and professions allied to medicine.

5. FAMILY JUSTICE SYSTEM

- **The Family Justice System** is a network of organizations including **family courts, the Children and Family Court Advisory and Support Service (CAFCASS), the Child Support Agency, and lawyers**.
- Safeguarding children's welfare is a key consideration for all professionals working in the Family Justice System. In all cases the child's welfare is the court's paramount consideration and the role of the court is to make decisions which are in the best interest of children based on the evidence before it and the law.

CHAPTER 11. FEVER IN ALL AGE GROUPS**I. ASSESSMENT TOOL FOR THE FEBRILE CHILD**

- All febrile patients with **green features** should have a **urine checked** and be sent home with an advice sheet on fever. If any **amber features** and no diagnosis reached **admit**.
- If any **red features** refer child **urgently** to a paediatric registrar.
- All febrile babies under 6 month are red light. i.e urgent

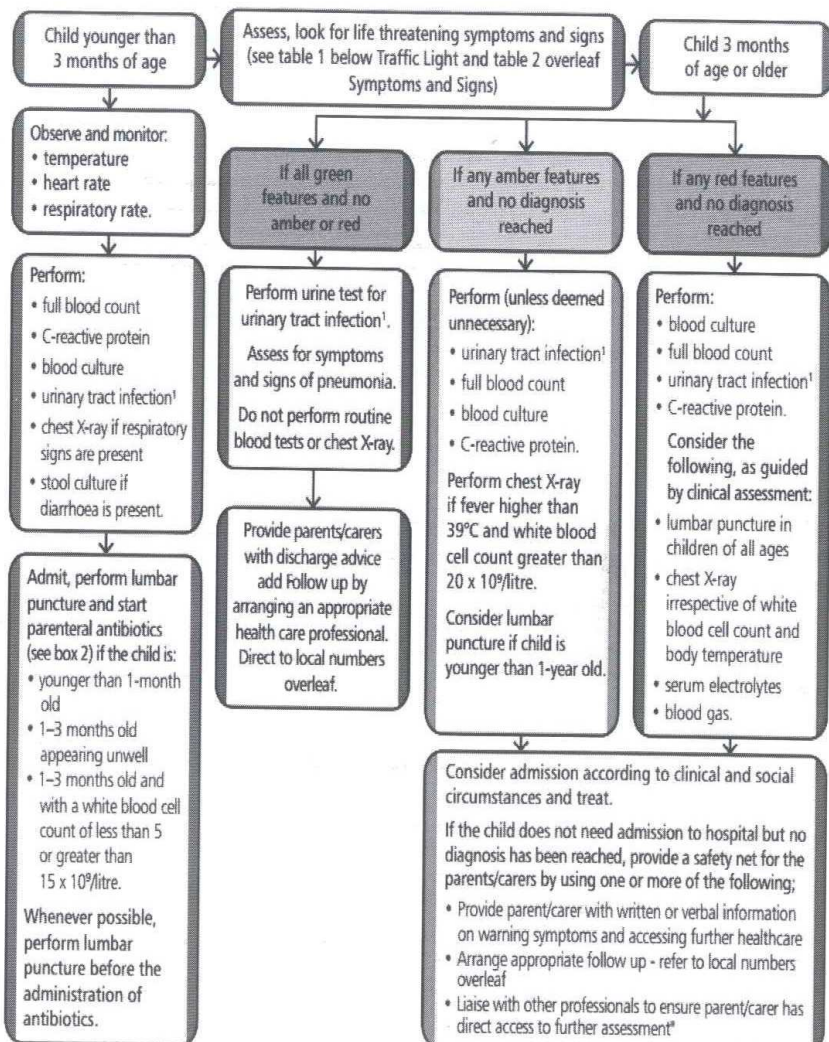
NICE TRAFFIC LIGHT SYSTEM

TRAFFIC LIGHT ALERT FOR FEBRILE CHILDREN IN ED			
GREEN-LOW RISK check urine and give fever advice sheet		AMBER INTERMEDIATE RISK- admit	RED – HIGH RISK OF LIFE THREATENING ILLNESS
Colour	Normal colour of skin, lips and tongue	Pallor reported by parent/carer	Pale/mottled/ashen/blue
Activity	Responds normally to social cues Content/smiles Stays awake or awakens quickly Strong normal cry/not crying	Not responding normally to social cues Wakes only with prolonged stimulation Decreased activity No smile	No response to social cues Appears ill to a healthcare professional Does not wake or if roused does not stay awake Weak, high-pitched or continuous cry
Respiratory		Nasal flaring RR > 50, age 6–12 mo RR > 40, age > 12 mo O ₂ sat ≤ 95% in air Crackles	Grunting RR > 60 /minute Moderate or severe chest indrawing
Hydration	Normal skin and eyes Moist mucous membranes	SBP >160 (<12 months), SBP >150 (12-24 months) SBP >140 (24-60 months) Dry mucous membranes Poor feeding in infants CRT ≥ 3 seconds Reduced urine output	Reduced skin turgor
Other	None of the amber or red symptoms or signs	Fever for ≥ 5 days**	Age 0–3 months, temperature ≥ 38 °C Age 3–6 months, temperature ≥ 39 °C Non-blanching rash Bulging fontanelle Neck stiffness Status epilepticus Focal neurological signs Focal seizures Bile-stained vomiting

* Swelling of a limb, non-weight bearing, not using an extremity and a new lump >2cm

Clinical Assessment Tool for the Febrile Child 0-5 Years

Management by a paediatric practitioner



¹ See 'Urinary tract infection in children', NICE clinical guideline CG54.

II. FEBRILE CONVULSION

DEFINITIONS

- A seizure associated with fever occurring in a young child.
- Most occur between **18 months and 3 years of age**.
- Febrile seizure is rare before **6 months** and after **6 years**.
- **Simple febrile convulsion** is an Isolated, generalized tonic-clonic seizure lasting less than 15 minutes, which does not recur within 24 hours or within the same febrile illness.
- **Complex febrile seizures**, have one or more of the following features: a partial (focal) onset or focal feature during the seizure; duration of more than 15 minutes; recurrence within 24 hours, or within the same febrile illness; incomplete recovery within 1 hour.
- **Febrile status epilepticus** is a febrile seizure lasting longer than 30 minutes.
- Febrile convulsions arise most commonly from infection or inflammation outside the CNS in a child who is otherwise neurologically normal.
- Most febrile convulsions will have ceased by arrival in the ED. The child should initially be assessed in an ABCDE manner. The history and examination should aim to determine the cause of the fever (e.g. **viral upper respiratory tract infection, otitis media, urinary tract infection**, etc.).
- The above NICE traffic light system is helpful to identify the likelihood of serious underlying illness.
- If the child is still fitting on arrival in the ED they should be managed using the status epilepticus guidance (See below).
- **DIAGNOSTIC APPROACH**
 - **Resuscitation:** The priority is to stop the fit and to stabilise the patient following standard APLS guidelines.
 - **Don't ever forget Glucose (DEFG!)** - check BM in ALL.
- **History**
 - The state of health prior to the fit: typically, the child is a little off colour or well prior to the fit.
 - Features of the fit:
 - Obtain an accurate description of the fit if possible
 - Importantly - whether consciousness was lost.
 - The eyes may roll up, the limbs may stiffen, there may be cyanosis, there may be generalised movements of either upper and/or lower limbs.
 - Previous medical history:
 - Particularly any previous history of fits.
 - Contact with infectious diseases.
 - Foreign travel.
 - Family history of epilepsy or of febrile convulsions.
 - Medication.
- **Assessment**
 - The unconscious child should be assessed in accordance with APLS guidelines.
 - Primary survey of ABCD, before a secondary survey general examination.
 - Continuous pulse oxymetry.
 - Investigation directed to the clinical findings, type of fit and age of the child.

DIFFERENTIAL DIAGNOSIS OF PAEDIATRIC SEIZURES

FIRST DAY OF LIFE	SECOND- THIRD DAY OF LIFE	DAY 4 TO 6 MONTHS OF AGE
Anoxia	Sepsis	Hypocalcemia
Hypoxia	Trauma	Infection
Trauma	Inborn errors of metabolism	Hyponatremia/hyponatremia
Intracranial hemorrhage	Hypoglycemia	Drug withdrawal
Drugs	Hypo/calcemia/magnesemia	Inborn errors of metabolism
Infection	Hyponatremia/hyponatremia	Hyperphosphatemia
Hypo/hyperglycemia	Hyperphosphatemia	Congenital anomalies
Pyridoxine deficiency	Drug withdrawal	Hypertension
	Congenital anomalies	

ED MANAGEMENT OF FEBRILE CONVULSION

• Children less than 6 months old

- Treat with caution (Any child under 6 months old with a high fever)
- By definition this is not a febrile seizure (rare before 6 months and after 6-year-old)
- Assume is CNS infection until proven otherwise.
- All are treated as for **meningitis**.
- **Antibiotic therapy** must be commenced
- Urgent discussion with the ED Duty doctor and on call paediatrician
- Antibiotic treatment should not be delayed whilst the septic screen samples are collected.

• Age 6 - 18 months

- Treated with extreme caution
- Signs of serious infection are few
- If severely unwell a full septic screen should be carried out after appropriate resuscitative measures have been taken.
- Antibiotic therapy should not be delayed if obtaining these samples proves difficult.
- If mildly or moderately unwell, the child should be observed closely and the following investigations should be performed
- **Laboratory:** Urine microscopy, Glucose, FBC, CRP, U&E, Calcium and Mg
- Further investigation should be guided by continuing clinical review.
- A full septic screen should be considered if the child fails to show clinical improvement after appropriate healing measures, particularly if any of the following features are present:
 - *The child looks toxic or is irritable*
 - *The child shows any sign of meningism*
 - *The child shows signs of drowsiness or delayed recovery from the fit*
 - *The fit is complex.*
- Refer to the Paeds Doctor on duty

• Age over 18 months

- Other children are easier to assess clinically.
- If the child is severely unwell, investigations should be carried out as for the severely unwell child under 18 months of age.
- If mildly or moderately unwell, clinical assessment is of the greatest importance.
- *Where there is an obvious source of infection, after thorough clinical assessment, no further investigations are required.*

- Where the source of infection is not obvious or the fit was complex then proceed as per above investigations.

DISPOSAL

- Following a febrile fit, it may be reasonable to send the child home if the following criteria are met;
 - Age > 1 year
 - The fit was simple
 - The child has fully recovered
 - There is an obvious source of infection
 - The child is not severely unwell
 - The parents are not unduly anxious
 - The child has had a previous febrile convulsion or there is a family history of febrile convulsions.
- As a general rule, cases involving first febrile fits should be discussed with Senior ED staff or with the duty paediatrician.
- **Indications** for referral to the paediatric team after a febrile convulsion include:
 - Children aged <18 months.
 - Signs of meningism.
 - Parental anxiety.
 - Complex or prolonged seizures
 - Systemically unwell.
 - Current or recent antibiotic use.
 - No clear focus of infection.
 - First febrile convulsion
- All children presenting with a febrile convulsion who are less than 1 year of age should be admitted.
- Between 1 year and 18 months of age cases should be discussed with the duty admitting paediatrician
- **DISCHARGE CHECKLIST**
 - If the child is to be discharged from the Emergency department check:
 - Appropriate treatment for the infection (if any)?
 - Advice about keeping the child comfortable (remove clothing, Paracetamol 15mg per kg every 4-6 hours orally and/or Ibuprofen syrup 5mg per kg every 8 hours).
 - An advice sheet should be given about febrile convulsions.
 - Follow-up - should be arranged within 24-48 hours (normally with the General Practitioner but occasionally by return for ED senior review).
- **OUTCOME & PROGNOSIS**
 - The parents should be counselled fully (nearly all parents think that their child is dying during the first febrile fit). The recurrence risk is less than 30% (1 in 6 have 3 fits or more). Most recurrences occur within one year of the first convulsion.
 - Often a strong family history, so siblings should be kept cool during illnesses.
 - Simple febrile fits have no relationship to the development of epilepsy (if the convulsion was complex then outpatient follow-up with an EEG and/or CT scan is indicated).
 - From a health prevention perspective, it is important to emphasise that the immunisation schedule should not be changed because of a simple febrile convulsion.

III. MENINGITIS & MENINGOCOCCAL SEPTICAEMIA

INTRODUCTION

- The diagnosis of meningococcal disease in its early stages, particularly in the first 4-8 hours, is often difficult owing to non-specific symptoms.
- A high clinical suspicion is required when assessing the unwell child in the ED.
- Deterioration is often rapid and irreversible unless appropriate management is instituted at presentation.
- Recommended standard pre-hospital treatment for suspected cases is **IM benzylpenicillin**.

DEFINITION

- **Meningococcal meningitis:** Inflammation of the pia and arachnoid mater, resulting from meningococcal
- **Meningococcal septicaemia:** Bacterial infection of the bloodstream by *Neisseria meningitidis* with subsequent bacterial endotoxin release, and rapid progression to shock and circulatory collapse.
- The rate of confirmed meningococcal disease is highest in **the under-fives**, particularly among infants.
- Case fatality ratios in children are highest in teenagers older than 14, followed by infants less than 1 year of age.
- There is a marked seasonal variation in incidence, with case numbers peaking in the winter months and during outbreaks of viral respiratory infection.
- Approximately 10% of the population are asymptomatic carriers in the nasopharynx.
- Carriage rates vary from 2% in children < 5 years and peak to 25% in children between the ages of 15-19 years.
- The most common serogroups that cause disease are **A, B, C, W125, Y**
- Since the introduction of vaccines to control Hib, serogroup C meningococcus and pneumococcal disease, serogroup B meningococcus is now the most common cause of bacterial meningitis and septicaemia in children.

CLINICAL ASSESSMENT

- Most children with early (4-8 hours) meningococcal septicaemia or meningitis present as an **acutely febrile child**.
- They may not have any other classical features of meningococcal septicaemia or meningitis (particularly in < 2 year of age).
- Other early symptoms may include *irritability, lethargy, vomiting, respiratory symptoms, cold hands and feet and myalgias (leg pains)*.
- *Presentations of meningococcal infection can be as meningitis alone (15%), septicaemia (25%), septicaemia and meningitis (60%), arthritis, osteomyelitis, conjunctivitis, endophthalmitis, chronic meningococcaemia.*
- Any sick child presenting to the ED should have baseline observations performed on arrival.
- These include heart rate, respiratory rate, BP/CRT, and conscious level (AVPU).
- The child should have a rapid 'Airway, Breathing, Circulation, Disability, Exposure, assessment, followed by a more detailed review.
- Any deviation from the normal range in combination with any of the following features should prompt consideration of a diagnosis of invasive meningococcal disease.

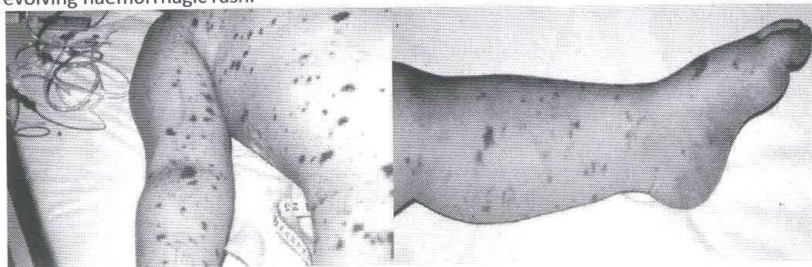
- **Presenting Features:**

FEATURES SUGGESTIVE OF MENINGOCOCCAL SEPTICAEMIA	FEATURES SUGGESTIVE OF MENINGOCOCCAL MENINGITIS
Early features: <ul style="list-style-type: none"> • Fever • Cold hands and feet • Pallor • Skin mottling • Extremity pain 	Early features: <ul style="list-style-type: none"> • Fever • Irritability • Lethargy • Respiratory symptoms • Vomiting
Late features: <ul style="list-style-type: none"> • Clinical shock • Confusion • Reduced consciousness • Neck stiffness • Rapid change in condition • Petechial rash 	Late features: <ul style="list-style-type: none"> • Bulging fontanelle • Headache • Altered mental state • Neck stiffness • Photophobia • Petechial rash

- In general, meningococcal disease is less likely if the disease has lasted more than 24 hours.

RASH IN MENINGOCOCCAL DISEASE

- Most patients with meningococcal septicaemia **develop a rash**.
- However, this will not always be a feature at initial presentation.
- The rash can range from scanty blanching macular or maculopapular lesions to a rapidly evolving haemorrhagic rash.



- The text-book **non-blanching rash** may be a very late sign, and the underlying meningitis or septicaemia is often very advanced by the time this rash appears.
- A generalised **petechial rash or purpuric rash** in any location, **in an ill child**, are strongly suggestive of meningococcal septicaemia and should prompt urgent treatment.

INVESTIGATIONS

- Blood glucose, FBC, Coagulation (PT), U&E, CMP
- ABG or capillary gas (acidosis/base deficit, bicarbonate, lactate)
- **Blood cultures** and **Meningococcal PCR** to aid in reaching a definitive laboratory diagnosis.
- **Lumbar puncture**
 - The collection of CSF should not delay the institution of antibiotic therapy.
 - PCR on CSF can still yield a positive result in samples collected after the start of treatment.

NICE CLINICAL GUIDELINE 102

INVESTIGATION/MANAGEMENT IN CHILDREN AND YOUNG PEOPLE WITH PETECHIAL RASH

- Give intravenous **ceftriaxone immediately** to children and young people with a petechial rash if any of the following occur at any point during the assessment (these children are at high risk of having meningococcal disease):
 - *Petechiae start to spread*
 - *The rash becomes purpuric*
 - *There are signs of bacterial meningitis.*
 - *There are signs of meningococcal septicaemia.*
 - *The child or young person appears ill to a healthcare professional.*
- If a child or young person has **an unexplained petechial rash and fever** (or history of fever) carry out the following investigations:
 - *Full blood count*
 - *C-reactive protein (CRP)*
 - *Coagulation screen*
 - *Blood culture*
 - *Whole-blood polymerase chain reaction (PCR) for N. meningitidis*
 - *Blood glucose*
 - *Blood gas.*
- In a child or young person with an unexplained petechial rash and fever (or history of fever) but none of the high-risk clinical manifestations:
- Treat with **IV ceftriaxone immediately** if the **CRP and/or white blood cell count is raised**, as this indicates an increased risk of having meningococcal disease.
- *Be aware that while a normal CRP and normal white blood cell count mean meningococcal disease is less likely, they do not rule it out. The CRP may be normal and the white blood cell count normal or low even in severe meningococcal disease.*
- Assess clinical progress by monitoring vital signs (respiratory rate, heart rate, blood pressure, conscious level [Glasgow Coma Scale and/or APVU], temperature), capillary refill time, and oxygen saturations. Carry out observations at least hourly over the next 4–6 hours. If doubt remains, treat with antibiotics and admit to hospital.
- If the child or young person is assessed as being at low risk of meningococcal disease and is discharged after initial observation, advise parents or carers to return to hospital if the child or young person appears ill to them.
- Be aware that in children and young people who present with a non-spreading petechial rash without fever (or history of fever) who do not appear ill to a healthcare professional, meningococcal disease is unlikely, especially if the rash has been present for more than 24 hours. In such cases consider:
 - *Other possible diagnoses*
 - *Performing a FBC and coagulation screen.*

INVESTIGATION/ MANAGEMENT IN CHILDREN WITH SUSPECTED BACTERIAL MENINGITIS

- In children and young people with suspected bacterial meningitis, perform a **CRP and white blood cell count**:
- If the CRP and/or white blood cell count is raised and there is a non-specifically abnormal cerebrospinal fluid (CSF) (for example consistent with viral meningitis), **treat as bacterial meningitis**. Be aware that a normal CRP and white blood cell count does not rule out bacterial meningitis.

- Regardless of the CRP and white blood cell count, if no CSF is available for examination or if the CSF findings are uninterpretable, manage as if the diagnosis of meningitis is confirmed.

MANAGEMENT OF MENINGITIS IN THE ED

General Management

- All children with suspected Meningitis in the ED should be managed in the presence of full resuscitation facilities, with continuous cardiac and oxygen saturation monitoring.
- A senior member of the ED medical team and the duty paediatrician should be involved at an early stage.
- **A & B:**
 - **Oxygen** should be administered to all patients, and an anaesthetist immediately involved if there is any concern about the child's ability to maintain their own airway and adequately self-ventilate.
 - Consider **Intubation** if GMSPS ≥ 8 or child requires **>40ml/Kg of fluid**
- **C:**
 - **Two wide bore IV access (or IO)**
 - Collect appropriate Blood tests
 - **Fluids**
 - 0.9% Normal Saline **20 mls/kg** is recommended for the first fluid bolus
 - 0.9% Normal saline or 4.5% human albumen for subsequent boluses
 - Repeated fluid boluses can be administered as needed, with the goal of attaining normal perfusion. If the child requires greater than **40 ml/kg** initial fluid resuscitation, or if they score **equal to or greater than 8** on the GMSPS (where used), it is important to consult with the local paediatric intensive care unit to consider elective intubation and ventilation, and rapid escalation of treatment with inotropic support and fluid management. In children suspected to have raised ICP secondary to meningitis, control the PaCO₂ within the normal range (4-4.5 kPa).
 - All children requiring intensive care support should have **central venous access** and an **arterial line**, NGT and **urinary catheter** inserted.
- **Drugs:**
 - **Antibiotics:**
 - The NICE guideline recommends **CEFTRIAXONE 80 mg/kg OD in > 3 month olds**.
 - **IV cefotaxime (50 mg/kg)** should be used as initial treatment of previously well children over 3 months with a diagnosis of IMD.
 - **If < 3 month olds: Cefotaxime + Ampicillin or Amoxicillin** (an antibiotic active against listeria).

1. CONFIRMED BACTERIAL MENINGITIS

- **Children and young people aged 3 months or older**
 - Treat ***H influenzae* type b meningitis** with **intravenous ceftriaxone** for **10 days** in total unless directed otherwise by the results of antibiotic sensitivities.
 - Treat ***S pneumoniae* meningitis** with **intravenous ceftriaxone** for **14 days** in total unless directed otherwise by the results of antibiotic sensitivities.
- **Children younger than 3 months**
 - Treat **Group B streptococcal meningitis** with **intravenous cefotaxime** for at least **14 days**. If the clinical course is complicated consider extending the duration of treatment and consulting an expert in paediatric infectious diseases.

- Treat bacterial meningitis due to *L. monocytogenes* with **intravenous amoxicillin or ampicillin for 21 days in total, plus gentamicin for at least the first 7 days.**
- Treat bacterial meningitis due to **Gram-negative bacilli** with **intravenous cefotaxime for at least 21 days** unless directed otherwise by the results of antibiotic sensitivities. If the clinical course is complicated consider extending the duration of treatment and consulting an expert in paediatric infectious diseases.

2. UNCONFIRMED BACTERIAL MENINGITIS

- In children and young people aged 3 months or older with unconfirmed, uncomplicated but clinically suspected bacterial meningitis, treat with intravenous **ceftriaxone for at least 10 days** depending on symptoms and signs and course of the illness.
- In children younger than 3 months with unconfirmed but clinically suspected bacterial meningitis, treat with **cefotaxime plus either ampicillin or amoxicillin for at least 14 days.**
- **Dexamethasone 0.15 mg/kg qds for 4/7** is recommended in bacterial meningitis of unknown aetiology and has been associated with improved neurological outcome.
- This should be commenced with or within 12-24 hours of the first antibiotic dose.

3. MENINGOCOCCAL DISEASE

- In children and young people with confirmed meningococcal disease, treat with intravenous **ceftriaxone for 7 days** in total unless directed otherwise by the results of antibiotic sensitivities.
- In children and young people with unconfirmed but clinically suspected meningococcal disease, treat with **intravenous ceftriaxone for 7 days in total.**
- In children and young people with suspected or confirmed meningococcal septicaemia, anticipate, monitor and correct the following metabolic disturbances using local or national protocols:
 - *Hypoglycaemia/ Acidosis/ Anaemia/ Coagulopathy.*
 - *Hypokalaemia/ Hypocalcaemia/ Hypomagnesaemia*

4. SEPTICAEMIA WITH SHOCK

- **INOTROPES**
 - The peripheral inotrope of choice is **Dopamine started at 10 mcg/Kg/min.**
 - **Adrenaline infusion** should be commenced if there is ongoing haemodynamic instability requiring large volume fluid resuscitation and escalation in inotrope dose. This should be given centrally or intraosseously.
- **COAGULOPATHY**
 - Give **10 ml/Kg FFP or 5 ml/Kg cryoprecipitate** if fibrinogen less than 1 g/dL.
 - Consider **transfusing packed red cells to maintain Hb >10 g/dL.**
- **STEROIDS**
 - Corticosteroids are only indicated in meningococcal septicaemia **with shock refractory to inotropes at 60 minutes**, when **carefully titrated hydrocortisone** may be considered to cover for absolute adrenal insufficiency.

5. MENINGITIS WITH RAISED ICP

- This will require paediatric neuro-intensive care.
- Ensure normocapnia (PaCO₂ 4-4.5 kPa) and ensure adequate oxygenation.
- For acute raised ICP, diuretics (**Mannitol 0.25 g/kg over 5mins**, followed by **frusemide 1 mg/kg**) are recommended, and should be repeated if clinically indicated.
- Overaggressive fluid resuscitation in raised ICP will exacerbate cerebral oedema.

- o Only children who are hypotensive secondary to septic shock require aggressive fluid replacement and inotropes to maintain cerebral perfusion pressure.
- o The patient should be nursed with **their head elevated** as much as possible in the midline position, with regular monitoring of pupil size and reactivity.
- o Keep normothermic, and treat seizures aggressively.

• CLINICAL FEATURES OF MENINGITIS WITHOUT SHOCK OR SIGNS OF RAISED ICP

- o Children with clinical features of suggestive of meningitis, but in the absence of shock or features of raised ICP should be treated empirically with:
 - **IV Cefotaxime**, and closely monitored for any signs of disease progression.
 - **Dexamethasone 0.4 mg/kg** administration as an adjunct to antibiotic therapy has been suggested to reduce neurological sequelae and hearing loss in children with meningitis, particularly if the pathogen proves to be H. Influenzae or S. Pneumonia.

6. MENINGITIS WITH RAISED ICP AND SEIZURES

- o Stepwise management of seizures:
 - **IV lorazepam 0.1 mg/kg or Midazolam IV or buccal (0.5 mg/kg)**
 - **Paraldehyde 0.4 ml/kg PR**
 - **Phenytoin infusion 18 mg/kg over 30 mins** with ECG monitoring
 - For persistent seizures **Thiopentone 4 mg/kg** (intubated patients only), or midazolam/thiopentone infusions should be considered with paediatric neuro-intensive care advice.

7. PROPHYLAXIS

- **Prophylaxis** should be given **within 24 hours** of diagnosis to:
 - o **Household members** who have had prolonged close contact within 7 days before the onset illness (also consider child minders who may be looking after the child for a number of hours, pupils in a same dormitory)
 - o **Kissing contacts** i.e. boyfriends/girlfriends
 - o **Healthcare workers** who have had direct exposure to droplets/respiratory secretions prior to completion of 24 hours of antibiotics (not required in nurseries/schools with isolated cases unless close contact)
- **Dosage:**
 - o **Rifampicin 600 mg every 12 hours for 2 days;**
 - o **Children Rifampicin 10 mg/kg bd for 2 days;**
 - o **Infants Rifampicin 5 mg/kg bd for 2 days**
 - o Other alternatives for adults are **ciprofloxacin** and **ceftriaxone**.

CHAPTER 12. APPROACH TO FITTING CHILD

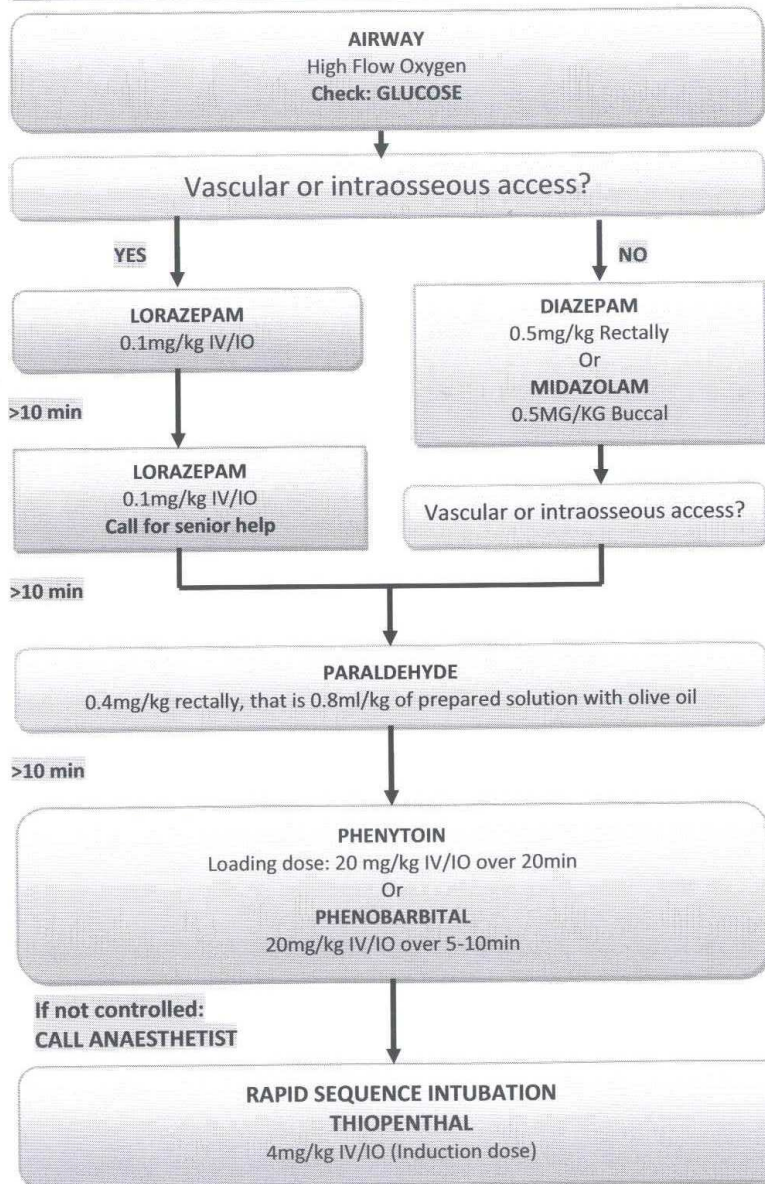
I. PAEDIATRIC STATUS EPILEPTICUS

INITIAL MANAGEMENT

- **A:** Ensure patent airway (position, suction, adjuncts, BMV)
- **B:** 100% O₂
- **C:** Check **glucose**, IV or IO access if no delay (otherwise PR Diazepam to stop fit after checking BM)
- **D:**
 - STOP THE FITTING (Algorithm)
 - Tepid sponging and Paracetamol if fever
 - Cefotaxime, acyclovir and erythromycin if aetiology unclear
 - **No Lumbar Puncture if reduced level of consciousness**

APLS ALGORITHM

- The current APLS algorithm for the treatment of the convulsing child is as follows:
- **Step 1 (5 minutes after start of convulsion):**
 - In a child that has been convulsing for 5 minutes or more an initial dose of benzodiazepine should be given:
 - **Lorazepam 0.1 mg/kg** should be given **IV or IO** if vascular access is available
 - **Buccal Midazolam 0.5 mg/kg** or **Rectal Diazepam 0.5 mg/kg** can be given as alternatives if no vascular access is available.
- **Step 2 (10 minutes after start of step 1):**
 - If the convulsion continues for a further 10 minutes a **second dose of benzodiazepine** should be given and **senior help should be summoned**.
- **Step 3 (10 minutes after start of step 2):**
 - At this stage senior help is needed to reassess the child and advise on management. The following management is recommended:
 - If not already on phenytoin then a **Phenytoin infusion 20 mg/kg IV infusion over 20 minutes should be set up**.
 - If already taking phenytoin then **phenobarbitone 20 mg/kg IV infusion over 20 minutes** can be used in its place.
 - **Rectal paraldehyde 0.8 ml/kg 50:50 mixture** whilst preparing the infusion can be considered.
- **Step 4 (20 minutes after start of step 3):**
 - If the child is still convulsing at this stage then an **anaesthetist** must be present and a rapid sequence induction with **thiopental** is recommended.

STATUS EPILEPTICUS- APLS ALGORITHM

CHAPTER 13. FLOPPY CHILD

Source: <http://www.emed.ie/Paediatrics/Miscellaneous>

• DIFFERENTIAL DIAGNOSIS OF AN ACUTELY FLOPPY CHILD "MIPHETS"

- **Metabolic:** Hypoglycaemia, Hypothermia, Hypercapnea, Renal Failure, Liver Failure
- **Intracranial infections:** Meningitis, Encephalitis, Malaria
- **Poisoning:** either accidental or deliberate.
- **Hypoxic ischaemic brain injury:** due to respiratory or circulatory failure (e.g. asthma, anaphylaxis, duct-dependent heart disease).
- **Epileptic seizures**
- **Traumatic brain injury:** Intracranial Haemorrhage, Cerebral Oedema
- **Sepsis**

BACKGROUND

- Occasionally infant presents to the ED who is described as "unwell" without any specific symptoms to guide the doctor.
- A judicious period of observation in the department will often be helpful (development of a pyrexia). Do not use a "baby check" scoring system for the assessment of such children (they may themselves be falsely reassuring).
- However, certain basic principles can be applied to the assessment of such children, and should be part of a routine examination:
 - *Is the infant smiling?*
 - *Does the infant play and reach for objects?*
 - *Does the child follow parent's eyes/fixes on objects etc?*
 - *Is the child feeding normally?*
 - *Has the child been wetting nappies normally?*
- If you, the nurses, (or the parents occasionally), assess that the answers to those questions are "yes", then the likelihood of a serious illness is small.
- However, this is not a substitute for full clinical assessment and investigations (where needed), and if there is any doubt the child should be admitted for observation.

I. THE UNWELL INFANT (<1-YEAR-OLD)

BACKGROUND

- This is one of the most difficult paediatric problems to deal with in the ED.
- You have to differentiate the basically well infant with a viral infection from the unwell with a more serious infection such as: Meningitis, UTI, LRTI...
- The following is a guide only on an approach to the assessment of these children.

ASSESSMENT

- Why has the parent brought the child into the ED?
- Evaluate the presenting symptoms, duration, what treatment has already been given.
- Has the child has its HIB vaccination in particular?

DOCUMENT

- Is the child smiling? Alert or listless? Miserable and irritable?
- Can they be easily consoled by the parent?
- Drinking well?
- Keeping down fluids? Normal number of wet nappies (4 a day rough minimum)?
- *If the answer to any of these is No then Beware!*

RAPID ASSESMENT

- **Airway and Breathing:**
 - Airway obstruction?
 - Increased work of breathing ... grunting, nasal flaring, recession, indrawing?
 - Respiratory rate and rhythm, Auscultation, Cyanosis?
- **Circulation:** Heart rate, Pulse volume, Capillary refill time, Skin temperature
- **Disability:**
 - Posture and tone, Pupils, Mental status - the AVPU scale
 - It should be possible to perform this assessment with the first minute.
 - If the child is very sick Call for help early.

DIAGNOSIS

- Head to toe exam - Does the child look well?
- Pyrexial? (some infants apyrexial even in septicaemia)
- Look for shin rashes -? viral
- Petechial rash of meningococcal septicaemia
- Note - Measles is very uncommon in the infants
- Dehydration: Sunken eyes, skin turgor, dry mucous membranes
- Peripheral circulation - well perfused? Capillary refill time prolonged (>2 sec)?
- Fontanelle - is it bulging or depressed
- Respiratory rate and pattern?
 - Normal infant is 30 - 40 breaths per minute
 - The ratio of pulse rate to respiratory rate is 4:1==>Raised in respiratory tract infection

INVESTIGATION

- Urine examination and culture for all/ Bag specimen, clean catch or SPA if trained.
- FBC: WBC unreliable in severe infection (often low), CRP unreliable in infants
- Check blood sugar - it may be low in sepsis. (DEFG).

INDICATIONS FOR ADMISSION

- According to diagnosis and assessment (as above) but admit any in whom you are unsure of the diagnosis.

MANAGEMENT

- **Pyrexial, irritable but well:**
 - Paracetamol & observation in ED - home if well
- **Unwell**
 - Paediatric/senior opinion
- **Very unwell with peripheral circulatory failure**
 - Call for help
 - ABC resuscitation (**Don't Ever Forget Glucose**)
 - Ceftriaxone and Penicillin before transfer
 - Dextrose if hypoglycaemic

RESUSCITATION

- Initial observations: Pulse, Resp, BP, Temp, Oxygen sats, Blood Glucose, Weight
- Initial investigations
- Secondary assessment
- Emergency treatment
- Definitive care

CHAPTER 14. GASTRO-INTESTINAL BLEEDING

I. UPPER GI BLEEDING

- The upper GI tract is considered any location proximal to the Ligament of Treitz (distal duodenum). The common manifestations are **hematemesis** or **melen**, while very brisk UGI bleeding can present with hemodynamic changes (symptoms of dizziness, dyspnea or shock) and/or hematochezia.
- The age of the pediatric patient is helpful when determining the differential diagnosis.

MOST COMMON ETIOLOGIES BY AGE	
Neonate	3 Years - 5Years
<ul style="list-style-type: none"> Maternal blood Hemorrhagic disease of the newborn Coagulopathies: Liver failure, DIC Gastritis: Stress, Sepsis, Protein Intolerance, Trauma (i.e. NG tube) Necrotizing Enterocolitis (NEC) 	<ul style="list-style-type: none"> Peptic Ulceration Gastritis: ASA, NSAIDs Varices Epistaxis Mallory-Weiss tear
1 Month - 1 Year	5+ Year
<ul style="list-style-type: none"> Peptic ulcer, Reflux esophagitis, Gastritis Foreign body Medications: ASA, NSAIDs Caustic Ingestion 	<ul style="list-style-type: none"> Peptic Ulceration Varices Coagulopathies: ITP, chemotherapy

• HISTORY

- Were there preceding complaints/signs of dyspepsia, dysphagia, abdominal pain, weight loss? What drugs have the patient taken recently that may contribute to gastritis or coagulopathy? Personal or family hx of easy bruising or bleeding?
- Jaundice or change in stool color may signify underlying liver dysfunction.
- A preceding choking bout may signify foreign body ingestion.
- Frequent epistaxis may indicate a nasopharyngeal source.

• DIAGNOSIS

- Is bleeding truly present? Red foods/liquids in the diet can resemble hematemesis.
- Perform **Gastrocult/Hemocult test** if unclear.
- Consider naso/oropharyngeal or respiratory sources of bleeding.
- A careful exam of the nares and oral pharynx should be done.
- The presence of "**coffee ground emesis**" represents blood altered by gastric contents and usually means that there has been slow bleeding from the region between the esophagus and the duodenum.
- Perform NG tube aspirate** if significant blood loss estimated (more than teaspoon).
- In addition to decreasing aspiration risk, this will aid in visualization via endoscope.
- Other characteristics of upper GI bleeding are **elevated BUN** and **hyperactive bowel sounds**, although these findings are not sensitive.
- Endoscopy** is the preferred diagnostic modality, and 90% of cases can be diagnosed if endoscopy performed **within the first 24 hours**.
- The most common causes have been identified as gastritis, esophagitis, duodenal ulcers, and esophageal varices.*
- Abdominal US** can assess portal HTN.
- Angiography** can be performed if endoscopy unsuccessful.

• ASSESSMENT OF THE PATIENT

- Hemodynamic stability is assessed by **vital signs**, which reflect the degree of blood loss.
 - Age-adjusted increased heart rate is always the first compensatory mechanism, while increased capillary refill, orthostatic hypotension, weakness/dizziness, and syncope are also signs.
- **Consider NG lavage** if bleeding is significant (>1 teaspoon)
- Labs: **FBC, Coags, U&E, LFTs**
- **Resuscitate** if hemodynamically unstable (see below)
- **Refer to Paediatric/Surgery**

• RESUSCITATION

- **Typing and cross matching of blood** should be done to be prepared if necessary.
- Fluid depletion should be corrected with **isotonic fluid** (IV/IO), as fast as necessary to reverse orthostatic hypotension. **Continuous monitoring of vital signs.**
- Hct is not a good measure of blood volume during acute hemorrhage.
- *If the bleeding is assessed to be severe, then the following should be considered: oxygenation, foley catheterization of the bladder, central venous line, transfusion of whole blood or PRBC, use of pharmacologic agents, intubation and ventilator support.*

II. LOWER GASTROINTESTINAL BLEEDING

- Although gastrointestinal bleeding is worrisome for parents, unlike adult medicine, it is rarely associated with malignancies in pediatrics.

CONFIRMING THE PRESENCE OF BLOOD IN THE STOOL

- **Hemoccult or Hematest.** This test material contains a peroxide which interacts with peroxidases in hemoglobin and causes a visible color change.
 - **False negatives** can be caused by large amounts of ascorbic acid in the diet or if intestinal bacterial degrade hemoglobin to porphyrin.
 - **False positives** can be caused by large amounts of rare red meat and certain vegetables: broccoli, cauliflower, turnips, radishes, and cantaloupe.
 - **Foods and Medicines** that can make stool appear bloody include red licorice, red pop, koolaid, jello, beets, iron and Pepto Bismol.

MOST COMMON ETIOLOGIES BY AGE

Neonate	1 month – 1 Years
<ul style="list-style-type: none"> ○ NEC- usually in preterm ○ Hirschsprung's disease associated with enterocolitis ○ Malrotation and associated volvulus ○ Swallowed blood- Do an Apt test to differentiate fetal from adult hemoglobin ○ Coagulaopathy 	<ul style="list-style-type: none"> ○ Anal or rectal fissures ○ Formula intolerance ○ Meckel's diverticulum ○ Hirschsprung's disease ○ Intussusception- most common in the ileocecal area ○ Lymphonodular hyperplasia ○ Infectious diarrhea/ HUS/ HSP
1 Year – 5 Years	5+ Year - Adolescence
<ul style="list-style-type: none"> ○ Polyps- may have large amount of bleeding and often pass spontaneously ○ Infectious diarrhea- either viral or bacterial 	<ul style="list-style-type: none"> ○ Similar to younger with the addition of ○ Inflammatory Bowel Disease

UPPER VS. LOWER INTESTINAL TRACT BLEEDING

- An important part of the work-up of GI bleeding involves differentiating upper from lower GI tract bleeding.
 - *If there is blood on the surface of the stool this is usually of anal-rectal origin*
 - *Bright red blood mixed in with stool usually is from below the ligament of Treitz but could be from above if bleed is brisk and large*
 - *Melana or tarry stools are usually above the ligament of Treitz*

EVALUATION OF BLEEDING

• HISTORY

- Amount of blood and appearance of stool. (Bright red blood vs. tarry stools)
- How long has there been bleeding?
- Associated symptoms of fever, weight loss, diarrhea, vomiting, constipation, pain
- Change of appetite, Diet
- Travel
- Family History
- Growth

• PHYSICAL EXAM

- Pallor
- Rashes, petechiae, purpura, hemangiomas, jaundice, telangiectasias
- Mouth lesions
- Abdominal exam for masses, tenderness
- Rectal exam
- Vital signs
- Jaundice (hepatic failure) or cutaneous bruising

EVALUATION

- The evaluation of the infant or child with blood in their stools is dependent on the history, general condition of the child, growth and development, amount of blood in the stool, the condition of the child including heart rate, blood pressure, amount of discomfort, and degree of anemia, if any.
- If necessary, the child should be stabilized.
- After a thorough history and physical exam, a **FBC, Reticulocyte count, smear, and Platelet count** should be performed.
- If the child is ill appearing, a **type and cross match** should be done.
- If the child is not ill and massive bleeding is not suspected, an **outpatient evaluation** may be performed.

CHAPTER 15. NEONATAL EMERGENCIES**OVERVIEW**

- Emergency department (ED) clinicians are challenged daily to recognize sick patients, diagnose life-threatening illness, and initiate treatment.
- Outside the everyday experience of managing sick patients in the ED are neonates; a specific group of patients who often present anxiety-provoking diagnostic challenges.
- Neonates often arrive at the ED with a nonspecific chief complaint or a history of symptoms that may or may not be benign.
- In order to recognize which neonates will require life-saving interventions, clinicians need to remain current on these life-threatening illnesses and their management.
- This clinical review addresses the recognition, diagnosis, and ED management of the more common neonatal emergencies.
- The mnemonic "**THE MIS FITS**" is helpful to quickly recall these critical diagnoses.

T- Trauma (nonaccidental and accidental)
H- Heart disease/Hypovolemia/Hypoxia
E- Endocrine (congenital adrenal hyperplasia, thyrotoxicosis)
M- Metabolic (electrolyte imbalance)
I- Inborn errors of metabolism: Metabolic emergencies
S- Sepsis (meningitis, pneumonia, urinary tract infection)
F- Formula mishaps (under- or overdilution)
I- Intestinal catastrophes (volvulus, intussusception, necrotizing enterocolitis)
T- Toxins/poisons
S- Seizures

I. TRAUMA (ACCIDENTAL AND NONACCIDENTAL)

- Emergency evaluation of neonates with head trauma may be a difficult process.
- An infant with nonaccidental head trauma may only have subtle historical findings and no physical exam findings. In addition, the presenting symptoms may be nonspecific.
- Early diagnosis of an occult head injury may prevent significant long-term morbidity.
- In fact, a recent study on abusive head injuries found that an apparent life-threatening event (ALTE) was often an unrecognized presenting symptom in infants. The study went on to recommend that infants with ALTE without an immediate obvious cause should be evaluated for head trauma with neuroimaging.
- Evaluation of neonates with a suspected injury should always include neuroimaging, which may include a **computed tomography (CT) scan, ultrasound, or magnetic resonance imaging (MRI)**.
- Skull x-rays are not always helpful because infants can have a significant intracranial injury without a skull fracture.
- **ED MANAGEMENT**
 - Will depend on presenting symptoms but should include evaluation and stabilization of the **ABC's** (airway, breathing, circulation), **DEFG** (bedside **blood glucose** evaluation), and appropriate **temperature regulation**.
 - If there is bruising or a known intracranial bleed, then the laboratory evaluation should include **FBC, Platelet count, PT and PTT**.
 - Neuroimaging should be completed after stabilization.

- The patient should be admitted and the injury **reported** to the appropriate department for abuse.
- A **skeletal survey and ophthalmologic exam** should be part of the hospital evaluation.

II. HEART DISEASE AND HYPOXIA

1. CYANOTIC HEART DISEASE

- Cyanosis is a pathologic process that requires immediate attention and evaluation.
- Although the differential diagnosis of cyanosis includes respiratory causes, infectious causes, central nervous system abnormalities and toxins, there is always the possibility of a cyanotic heart defect and so it should always be considered.
- Congenital heart defects that present with cyanosis are referred to "**Terrible 5T's**" and are listed below:
 - *Tetralogy of Fallot (TOF)*
 - *Tricuspid atresia (TA)*
 - *Transposition of the great vessels (TOGV)*
 - *Total anomalous pulmonary venous return (TAPVR)*
 - *Truncus arteriosus (TA)*
- Cyanotic heart defects may not be detected in the newborn nursery because there is still adequate oxygenated blood to the systemic circulation through a **PATENT DUCTUS ARTERIOSUS (PDA)**.
- Although the PDA typically **functionally** closes in the first 10-14 hours of life, several factors can delay its closure, including ("**PHAR**") **Prematurity, Hypoxia, Acidosis, and Respiratory distress**,
- The PDA is **anatomically** closed by **2 weeks of age**, contributing to the possibility of a relatively delayed detection of cyanotic heart defects.
- **Providing 100% oxygen** is helpful in differentiating between cardiac and noncardiac causes of cyanosis:
 - *After providing oxygen, a noncardiac disease should have at least a 10% increase in the pulse oximetry value, whereas cyanotic heart disease will have minimal change in the oxygen saturation.*
- This can also be confirmed with a **hyperoxia test**, which involves an initial arterial blood gas (ABG) on room air and then a repeat ABG after 10 minutes of 100% oxygen. There should be only minimal change in PaO₂ after 10 minutes of oxygen if the cause is cardiac.
- The ABG should include a methemoglobin percentage because congenital methemoglobinemia, severe diarrhea, and toxins can result in methemoglobin accumulation and cyanosis in a neonate. The management for **methemoglobinemia** would include **methylene blue 1-2 mg/kg IV over 5 minutes**.
- During stabilization, the physical exam should include blood pressures in all 4 extremities and a careful cardiac exam. Although a murmur may be audible, the absence does not exclude a cardiac defect.
- **INVESTIGATIONS**
 - ABG
 - CXR
 - ECG
 - Echocardiogram is diagnostic.

- **ED MANAGEMENT OF CYANOTIC HEART DISEASE**

- Prostaglandin E1 (PGE1) as a bolus of 0.05 mcg/kg IV, followed by an infusion of 0.05-0.01 mcg/kg/min IV.
- Intubation may not be necessary but equipment to secure the airway should be readily available, as a non-dose-dependent complication of PGE1 is profound apnea.
- Also, patients who require transport to a facility for pediatric subspecialty care may require definitive airway management prior to transportation.

2. ACYANOTIC HEART DISEASE

- Acyanotic heart disease typically presents with symptoms of **congestive heart failure**.
- There is usually a more gradual clinical decompensation when compared with the cyanotic heart defects and it may not present until after the first **2-3 weeks of age**.
- **Causes of Congestive Heart Failure in Neonates**
 - *Acyanotic heart disease*
 - Ventricular septal defect (VSD),
 - Atrial septal defect (ASD),
 - Patent ductus arteriosus (PDA),
 - Coarctation of the aorta
 - Severe anaemia/ Trauma/ Sepsis
 - Supraventricular tachycardia (SVT)
 - Metabolic abnormalities/ Thyrotoxicosis
 - Systemic lupus erythematosus (SLE)
- The classic symptoms for congestive heart failure include tachypnea, tachycardia, and hepatomegaly. The history may include poor or slow feeding, sweating or color change with feeding, and poor weight gain.
- **INVESTIGATIONS**
 - FBC, U&E, CXR, ECG.
 - An echocardiogram will be diagnostic of the heart defect.
- **ED MANAGEMENT**
 - Initial management will include stabilization of the ABC's
 - Furosemide 1mg/kg IV, plus Dopamine 5-15 mcg/kg/min IV or Dobutamine 2.5-15 mcg/kg/min IV for cardiovascular support.
 - Careful attention is necessary to not fluid-overload these patients.
 - Pediatric cardiology should be consulted.
 - A Pediatric cardiothoracic surgeon may be required for inpatient management.

3. SUPRAVENTRICULAR TACHYCARDIA

- Presenting complaints may range from tachycardia to poor feeding, irritability, heart failure, and shock. This is not usually a difficult diagnosis because the heart rate is sustained at **≥ 220 beats per minute with a QRS < 0.08 second**.
- ED management is dependent on the patient stability at presentation.
- **IN A STABLE PATIENT**
 - **Vagal maneuvers** at this age include icing the face, avoiding the nares.
 - If unsuccessful, IV access should be established, and adenosine **0.1 mg/kg IV push** followed immediately by flush should be administered (maximum of 6 mg/kg).
 - If SVT persists then a second dose of adenosine **0.2 mg/kg IV** (maximum of 12 mg/kg).
 - After a further two minutes, another dose of **300 mcg/kg adenosine** should be given

- If the child remains in stable SVT despite these measures then the guidelines recommend that following be considered:
 - **Adenosine 400-500 mcg/kg**
 - **Synchronous DC shock**
 - **Amiodarone**
- **AN UNSTABLE PATIENT**
 - Without IV access should be treated with **synchronized cardioversion (1 - 2 J/kg)**.
 - If there is established IV access and adenosine is readily available, then the initial cardioversion may be attempted pharmacologically.
 - If the SVT is unresponsive to adenosine or synchronized cardioversion or if a wide QRS is suspected, then **amiodarone 5 mg/kg IV over 20-60 minutes** may be administered.
 - Alternatively, **Procainamide 15 mg/kg IV over 30-60 minutes** may be administered.
 - *Amiodarone and Procainamide should not be administered together because the combination can lead to hypotension and widening of the QRS complex.*
 - **Lidocaine 1 mg/kg IV** is a final option for a wide QRS and should only be used in consultation with a pediatric cardiologist.
 - A 12-lead ECG should be obtained prior to and after conversion from SVT to normal sinus rhythm.
- This is a useful diagnostic tool for the cardiologists to help determine further management. A Pediatric Cardiologist should be consulted for further evaluation.

III. ENDOCRINE EMERGENCIES

1. CONGENITAL ADRENAL HYPERPLASIA

- Although these patients are often diagnosed at birth by routine newborn screening,
- Occasionally the diagnosis is missed because of an inadequate blood sample, laboratory error, or inability to contact the family. In this instance, the patient may present in the first few weeks of life with symptoms of vomiting, hypoglycemia, or even shock.
- The most common cause of congenital adrenal hyperplasia (CAH) is a **deficiency in the 21-hydroxylase enzyme**.
- Management includes **stabilization of the ABC's**, a **bedside blood glucose measurement**, and **serum electrolytes**.
- As with any presentation of a critically ill patient, sepsis should also be included in the differential and empirical treatment with antibiotics may be warranted. The electrolyte abnormalities may include **hyponatremia and hyperkalemia**.
- *Hypotension that is unresponsive to fluids or inotropes should heighten your suspicion of CAH.* The patient should be treated with **hydrocortisone 25-50 mg/Kg IV**.
- It is imperative to also treat the hypoglycemia.
- Often hyperkalemia in these patients will respond to fluid therapy; however, if the patient is symptomatic or has ECG changes, then calcium chloride, sodium bicarbonate, insulin and glucose, and sodium polystyrene sulfonate (*Kayexalate*) may be necessary.
- Refer for Pediatric critical care management and endocrine consultation.

2. THYROTOXICOSIS

- Neonatal thyrotoxicosis may develop in infants **born to mothers with Graves disease**.
- It is caused by transmission of maternal **thyroid-stimulating immunoglobulin**.

- The presentation is often delayed and may present to the ED with symptoms such as poor feeding, failure to thrive, tachycardia, irritability, hyperthermia, vomiting, diarrhea, jaundice, thrombocytopenia, respiratory distress, heart failure and shock.
- Initial diagnosis may be difficult without a clear history of Graves disease from the mother.
- Evaluation should include **thyroid functions tests**.
- Treatment after stabilization will include:
 - **Propranolol 0.25 mg/kg IV** for the tachycardia, and
 - **Propylthiouracil (PTU) 1.25 mg/kg IV** followed by **Lugol's solution 1-5 drops** by mouth.
- The Lugol's solution should be given **1 hour after the PTU**.
- This will help to control the hypermetabolic state.
- Endocrine consultation and admission to a pediatric hospital is recommended.

IV. SEPSIS

- It is standard of care to complete a full sepsis evaluation (FBC, blood culture, urinalysis, urine culture, cerebral spinal fluid [CSF] culture and analysis, and CXR) in any neonate with a **rectal temperature of $\geq 38^{\circ}\text{C}$** .
- Although the evaluation of a febrile neonate is well supported, it is the recognition of sepsis and the initiation of a work-up in neonates with a less specific presentation that is a more difficult challenge.
- The symptoms and historical facts that should prompt the consideration of a full sepsis evaluation include *poor feeding, irritability, apnea, hypothermia, jaundice, rashes, increased sleeping, seizures, or vomiting*.
- Initial laboratory screening is not always helpful in recognizing those neonates with an invasive bacterial infection. It has been demonstrated that the use of peripheral white blood cell count is not helpful to differentiate febrile neonates with a more serious bacterial infection from those without a serious bacterial infection.
- In addition, the urinalysis may also be unremarkable in those neonates with a culture positive UTI.
- It is standard of care to administer broad-spectrum antibiotics to all neonates who undergo a sepsis evaluation or present with life-threatening symptoms that do not have another readily apparent cause.
- **Recommended Antibiotics and Dosages for Neonatal Sepsis**
 - **Ampicillin** 50-100 mg/kg IV **and**
 - **Gentamicin** 2 mg/kg IV **or**
 - **Cefotaxime** 50-100 mg/kg IV
 - **Acyclovir** 20 mg/kg IV

1. NEONATAL HERPES

- Deserves specific mention because the symptoms may be subtle and there may be no maternal history in 60% to 80% of women with an unrecognized infection.
- Early recognition and treatment with **acyclovir 20 mg/kg IV** may significantly decrease the mortality from 90% to 31%.
- Initiation of treatment should be considered in any neonate with *high fever, CSF with a lymphocytosis or numerous red blood cells in an atraumatic spinal tap, seizures, or a known maternal history of herpes infection*.
- The CSF analysis should include a **Herpes PCR** and **Herpes Culture**.

- There may be an elevation on liver function tests and a CXR may demonstrate pneumonitis. These neonates typically require a higher level of care in a pediatric ICU.

2. CUTANEOUS CELLULITIS

- Should broaden the antibiotic coverage to include an antistaphylococcal agent such as **clindamycin 10 mg/kg IV**.
- **Omphalitis**, a periumbilical infection, often requires fluid resuscitation and prompt surgical intervention because of the possible extension to the peritoneum.
- These patients should also undergo a full sepsis work-up.

V. INTESTINAL CATASTROPHES

- Vomiting in the neonatal period should always prompt the consideration of a pathologic process. It may be difficult to differentiate a life-threatening cause from a mild viral gastroenteritis or even severe gastroesophageal reflux.
- The initial symptoms may be nonspecific and the history may not be helpful in a neonate who has not developed a normal pattern. Bilious emesis is always concerning and should always initiate a pediatric surgery consultation. (Refer to Chapter 7/ Abdominal Pain)

1. TOXIC MEGACOLON

- Toxic megacolon or enterocolitis is a life-threatening presentation of a patient with **Hirschsprung disease**. Hirschsprung disease occurs in 1 out of 5000 live births and may often go unrecognized because constipation is common and usually benign.
- The history of constipation, especially with the additional history of failure to pass meconium in the first 24 hours of life, should increase suspicion of Hirschsprung disease.
- Presenting symptoms may include poor feeding, vomiting, irritability, abdominal distention, and hematochezia and shock as the condition progresses to enterocolitis.
- Initial management should include **stabilization of the ABC's, fluid resuscitation, and administration of broad-spectrum antibiotics**.
- **An abdominal radiograph** may reveal an enlarged or dilated section of colon.
- Surgical consultation and pediatric critical care management is necessary in the presence of enterocolitis.

2. NECROTIZING ENTEROCOLITIS

- Although **neurotizing enterocolitis (NEC)** is classically a disease of premature neonates that is diagnosed in the neonatal intensive care unit, it may occasionally occur in the term neonate after discharge from the newborn nursery. These neonates may present with symptoms similar to those with Hirschsprung enterocolitis.
- **MANAGEMENT OF NEC:**
 - Stabilization of **ABC's, Fluid resuscitation, and Nasogastric tube placement**.
 - Administration of **broad-spectrum antibiotics**,
 - **A PFA** that demonstrates **pneumatosis intestinalis** or **portal air** is diagnostic of NEC.
 - Pediatric surgical consultation, and critical care management is required.

VI. HYPERBILIRUBINEMIA (JAUNDICE)

- Jaundice is a physical finding in the neonate that may represent a normal process in a healthy baby or a more severe or life-threatening illness.
- Because jaundice is a common complaint, it may be difficult to recognize which neonates require more attention.
- Initial evaluation will be dependent on the clinical presentation but should include laboratory evaluation for **conjugated (direct) and unconjugated (indirect) bilirubin, Hematocrit, Reticulocyte Count, and Coombs test.**
- **Direct hyperbilirubinemia** is always pathologic and the more common causes include **biliary atresia, alpha-1 anti-trypsin deficiency, and hepatitis.**
- **Indirect hyperbilirubinemia** is usually due to **breastfeeding or normal physiologic causes,** but the more concerning causes include **ABO incompatibility, sepsis, glucose-6-phosphate deficiency, spherocytosis, Gilbert's disease, or Crigler-Najjar syndrome.**
- ED management should include stabilization of the **ABC's, a bedside blood glucose level** if warranted, and laboratory evaluation as described above.
- **Initiation of phototherapy** or in more severe cases **exchange transfusion** -- is dependent on the **neonate's gestational age and total serum bilirubin.**
- Consultation with other pediatric subspecialists, including a pediatric gastroenterologist, may be necessary depending on the suspected etiology of the hyperbilirubinemia.
- **Recommendations for Phototherapy and Exchange Transfusion in the Healthy Term (>38Weeks) Neonate**

AGE	PHOTOTHERAPY	EXCHANGE
24 hours	12 g/dL	19 g/dL
48 hours	15 g/dL	22 g/dL
72 hours	18 g/dL	24 g/dL
> 96 hours	20 g/dL	25 g/dL

VII. TOXINS

- Toxic ingestions are uncommon in this age group, but occasionally result from a maternal ingestion in a breastfeeding mother, homeopathic remedies, or overuse of accepted medications.
- Although teething does not occur in the first month of life, colic is a common concern at this point and results in lost sleep and frustration.
- Teething gels may be used as an attempt to relieve distress for both parents and neonates.
- Note that teething gels often contain **benzocaine** which may cause methemoglobinemia with overuse.
- Star anis tea is a homeopathic remedy also used in several cultures for infantile colic.
- Finally, **baking soda** has been used for intestinal gas and has resulted in serious toxicity.
- **ED management**
 - Primarily supportive and will depend on the clinical presentation.
 - Hospitalization for monitoring and observation is recommended.

CHAPTER 16. PAIN MANAGEMENT IN CHILDREN

I. ASSESSMENT OF ACUTE PAIN IN CHILDREN

• BACKGROUND

- Pain is commonly under-recognised, under-treated and treatment may be delayed.
- Drug choice and dosage may also cause problems due to unfamiliarity.
- Recognition and alleviation of pain should be a priority when treating ill and injured children.





• Assessment of acute pain in children in the ED

- Children < 5 years: FLACC
- Children 5-7 years: Wong Baker FACES
- Children > 7 years: Use VAS (scale 0-10 [10 worse pain ever])

FLACC SCALE < 5 YEARS

FLACC SCALE (FACE, LEGS, ACTIVITY, CRY, CONSOLABILITY SCALE)			
	0	1	2
Face	No particular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking, or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid or jerking
Cry	No cry (awake or asleep)	Moans or whimpers; occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging or being talked to, distractable	Difficult to console or comfort

WONG BAKER FACES (5-7YO)

WBF	NO PAIN	MILD PAIN	MODERATE PAIN	SEVERE PAIN
Faces pain score				
	0	1 - 3	4 - 6	7 - 10
Behaviour	Normal activity No reduced movement Happy	Rubbing affected area Decreased movement Neutral expression Able to play / talk normally	Protective of affected area Decreased movement / quiet Complaining of pain Consolable crying Grimaces when affected part moved / touched	No movement or defensive of affected part Looking frightened Very quiet Restless, unsettled Complaining of lots of pain Inconsolable crying
Example	Bump on head	Abrasion / Small laceration Sprain ankle / knee # fingers / clavicle Sore throat	Small burn / scald Finger tip injury # forearm / elbow / ankle	Appendicitis Large burn # long bone / dislocation Appendicitis Sickle crisis

PAIN RULER**HOW TO TREAT PAIN**

- **Non-Pharmacological**

- **Psychological strategies:** involving parents, cuddles, child-friendly environment, and explanation with reassurance all help build trust.
- Also, distraction with toys, blowing bubbles, reading, or story-telling using superhero or magical imagery to make the pain go away
- Non-pharmacological adjuncts such as limb immobilisation, dressings for burns

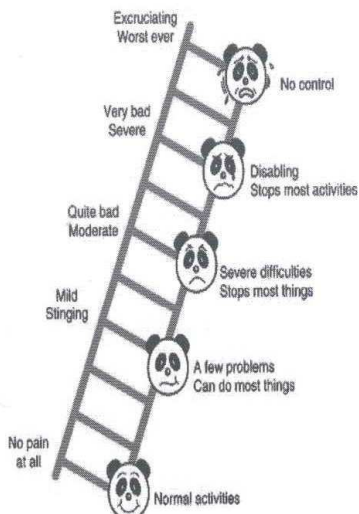
- **Pharmacological**

- **Pain management - Meds**

- Pharmacological agents, via a variety of routes: see attached algorithm.
- Use **TAC** in preference to EMLA for topical anaesthesia. For superficial wounds, topical anaesthesia should be used in preference to Lignocaine infiltration.
- Also, local or regional anaesthesia are useful (e.g. femoral and auricular blocks).
- For procedures, departments may consider conscious sedation using Ketamine (IV / IM) (more on Ketamine sedation). PO/IV/IN options include, Non-opioid, Opioid (including intra-nasally delivered Fentanyl) and inhaled (N₂O).

- **Contra-indications**

- **Ibuprofen / Diclofenac:** avoid if previous reactions to NSAIDs or in moderate or severe asthmatics.
- **Intravenous morphine:** use with caution if risk of depression of airway, breathing or circulation.
- **Oral and parenteral analgesia**

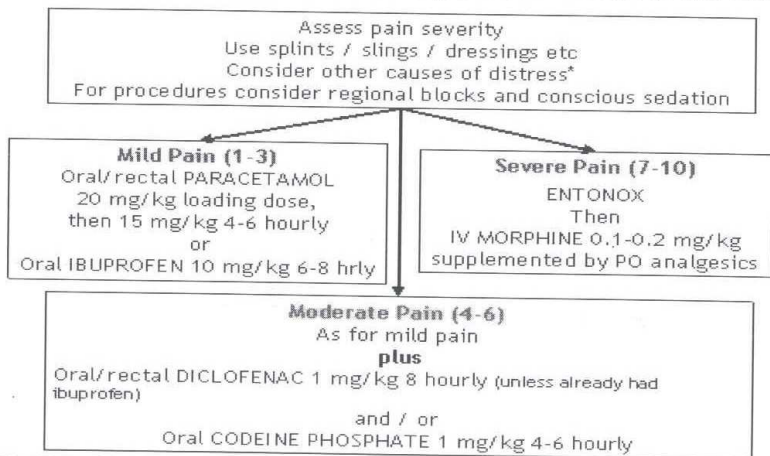


DRUG	DOSE PER KILO	TIMES A DAY
Paracetamol	15mg/Kg PO, Up to 30mg/kg PR	4-6 Hrly, Max 60mg/kg/day
Ibuprofen (beware in asthmatics)	10mg/Kg orally	QDS to max 20mg/Kg/24 hrs
Diclofenac	1mg/Kg PO or PR	TDS to max 3mg/Kg/day or 150mg daily
Codeine	1mg/Kg orally to max 30mg each dose	4 - 6 Hrly
Morphine	0.1mg/Kg IV 200 mcg/kg PO in <1yo 400 mcg/kg PO in 1-2yo	PRN (Reverse with Naloxone 10mcg/Kg then 100mcg/Kg)
Sucrose	>1500g child: 0.2-0.25ml of 24% Sucrose (Sweetease) dropped on the anterior tongue in divided doses 2 minutes before procedure, continue during procedure for a total dose of 2mls (action lasts approximately 5 minutes)	Maximum of 4 doses in 24 hours More on Sucrose
Fentanyl Intranasal	I.N. 1.5 mcg/kg/dose (use 50 mcrg/mL soln.)	

LOCAL ANAESTHETICS

- **Ametop**
 - o Can be used in children over 1-month old, Leave in situ for 30 mins for venepuncture
 - o Remove after 1 hour, Lasts 4 - 6 hours after removal
 - o Do not use on mucous membranes
- **Emla**
 - o Children over 1 year of age, should be left in situ for minimum 1 hour
 - o After 2 hours, will provide anaesthesia to 6 mm depth
- **Lignocaine 1%**
 - o Max 3mg/Kg
- **Adrenaline & cocaine gel**
 - o 1ml of gel per 1cm of wound, to max 4mls
 - o Not on mucous membranes or abrasions
 - o Controlled drug

Algorithm for treatment of acute pain in children in the ED



Journal of Intensive Care Medicine Feb 2008

II. KETAMINE SEDATION IN CHILDREN

- **INDICATIONS**
 - o Ketamine sedation is ideal for short, painful or frightening procedures.
 - o It may facilitate:
 - Suturing lacerations under local anaesthesia
 - Removal of foreign bodies
 - Orthopaedic procedures including joint relocation, fracture manipulation or joint aspiration
 - o Two important points to consider before opting for ketamine sedation:
 - **Ensure that the child has received appropriate analgesia:** This might include intranasal diamorphine, paracetamol, ibuprofen or all three as appropriate.
 - **Have alternative strategies been considered** e.g. Entonox or even more concentrated nitrous oxide/oxygen mixtures?

- **CONTRA-INDICATIONS**

- Prior adverse reaction to ketamine
- Age less than 12 months
- Active upper or lower respiratory tract infection
- Active asthma
- Unstable or abnormal airway: Tracheal surgery or stenosis.
- Proposed procedure within the mouth or pharynx
- Patients with severe psychological problems such as cognitive or motor delay or severe behavioural problems
- Previous psychotic illness/ Uncontrolled epilepsy/ Intra-ocular pathology
- Significant cardiac disease
- Recent significant head injury or reduced level of consciousness
- Intracranial hypertension with CSF obstruction
- Hyperthyroidism or thyroid medication
- Porphyrria
- A relative contra-indication that might result in a child receiving in-patient general anaesthesia is commonly **a lack of adequate ED resources**: typically, because of excess departmental workload.

- **Consent**

- Seek informed consent from the parent/guardian and older child, including in your discussion potential risks vs benefits, adverse events and alternative options.

- **Clinical adverse effects**

- Key adverse clinical effects that you should consider discussing are:
 - *Vomiting – 10%*
 - *Laryngospasm – 0.3%*
 - *Need for general anaesthetic (including suxamethonium) – 0.02%*
- Written consent for both the sedation and the procedure is advised.*

- **Dosage**

- **IV route: Ketamine IV 1 mg/kg slowly** (no less than a minute) so as to avoid apnoea.
- Within 60 seconds you should sense that the child becomes vacant, demonstrating occasional nystagmus.
- Supplemental (slow) **IV doses of 0.5 mg/kg** may be required should you deem the level of sedation inadequate, or if the procedure is prolonged.
- **IM route:** An alternative strategy is **2.5 mg/kg IM injection in the lateral aspect of a thigh** (prepared with topical local anaesthetic if time allows).
- Expect to wait 5 – 8 minutes for clinical effect.
- Use top-up doses of **1 mg/kg IM** as required.

- **Post-procedure**

- **College of Emergency Medicine post-procedure advice:**
 - The child should recover in a quiet, observed and monitored area under the continuous observation of a trained member of staff.
 - Recovery should be complete between **60 and 120 minutes**, depending on the dose and route used.
 - The child can be safely discharged once they are able to ambulate and vocalise/converse at pre-sedation levels.
 - An advice sheet should be given to the parent or guardian advising rest, quiet and supervised activity for the remainder of that day.

- The child should not eat or drink for two hours after discharge because of the risk of nausea and vomiting.

Key Learning Points

- Ensure that your child has received appropriate analgesia before considering sedation
- Carefully consider alternative strategies to sedation in general
- The College guideline allows you to proceed with ketamine sedation for non-fasted children, if the clinical need dictates urgent action
- Specific contraindications to Ketamine sedation include infants and children with **URTIs** or **significant learning difficulties**
- Slow IV Ketamine administration, no less than 60 seconds
- ECG, NIBP, RR, sats monitoring and supplemental oxygen are all advised
- Simple airway manoeuvres will counter most airway problems
- The need for suxamethonium in response to airway difficulty is rare
- A primary adverse event arising later than 30 minutes following the last Ketamine dose is exceptional
- There should be a documentation and audit system in place within a framework of clinical governance

III. PAEDIATRIC INTRANASAL FENTANYL

• BACKGROUND

- The intranasal (IN) route of medication administration has shown to be extremely effective in rapidly treating pain in children
- I.N. medications can be drawn up in a syringe and administered immediately using the MAD®: (Mucosal Atomiser Device) into one or two nostrils.
- The IN route can also be used when a patient is unable to take oral medication due to nausea and/or vomiting.
- Absorbed by the highly vascular mucosal membranes, the onset of action is considerably faster than the oral route as it does not require gastrointestinal absorption.
- Fentanyl is a potent opioid, approximately 100 times more potent than morphine, and with a shorter duration in action.
- Using the MAD®: in administering medication reduces the need for obtaining IV access.

• INDICATIONS

- *Burns.*
- *Painful wound dressing.*
- *Long bone fractures.*

• CONTRAINDICATIONS

- Head, chest or abdominal trauma
- Hypovolaemia
- Decreased level consciousness
- Allergy to Fentanyl
- Epistaxis
- Children under 1 year of age
- Precautions if child has blocked nose or URTI as there may be unreliable drug delivery.
- Any such patient should have their nasal cavity gently suctioned prior to administration.

CHAPTER 17. LIMPING CHILD**BACKGROUND**

- A limp is an asymmetrical gait caused by pain, weakness or deformity
- Limp is never normal and a cause must be established
- Limp must be considered in terms of the child's development

DIFFERENTIALS

- Toddler (1-3 years)
 - ✓ Toddler's fracture
 - ✓ DDH
 - ✓ Discitis
 - ✓ Cerebral palsy
- Child (4-10 years)
 - ✓ Transient synovitis
 - ✓ Perthes disease
- Adolescent
 - ✓ SUFE
 - ✓ Osgood-Schlatter disease
 - ✓ Osteochondritis dissecans
- All age groups
 - ✓ Septic arthritis
 - ✓ Osteomyelitis
 - ✓ Juvenile idiopathic arthritis (JIA)
 - ✓ Reactive arthritis
 - ✓ Malignancy
 - ✓ Non-accidental injury

PERTHES DISEASE

- Avascular necrosis of the femoral head
- Age 2-10 years, M>F
- Bilateral in 10-15%
- Gradual onset limp & pain
- Early diagnosis by MRI
- Most recover with bed rest, pain relief & physiotherapy
- Severe cases require surgical intervention

TRANSIENT SYNOVITIS

- Commonest cause of acute hip pain in children
- Age 2-10 years, M>F
- Preceded by viral infection
- Usually resolves within 1 week with reassurance, bed rest and simple analgesia

HISTORY

- Painful or painless
- Duration of symptoms
- Arthralgia (joint pain) or arthritis (joint pain & swelling & increased temperature)
- History of trauma
- History of fever/toxicity
- Birth history (breech, oligohydramnios)
- Family history (arthritis, DDH)

EXAMINATION

- Centiles & vital signs
- Observe gait
- Gower's test
- Measure limb length
- Full neurological and musculoskeletal exam
- Examine spine
- Examine skin for rash, bruising
- Palpate for lymphadenopathy

INVESTIGATIONS

- Bloods: FBC, CRP, ESR, blood film
- Blood culture
- X-ray affected joints
- U/S scan ± joint aspiration
- Bone scan or MRI

TREATMENT

- Depends on the diagnosis
- Conservative: rest, NSAIDs
- Broad spectrum IV antibiotics if septic arthritis or osteomyelitis
- Orthopaedic intervention if Perthes disease, SUFE, DDH

TAKE HOME MESSAGES

- The limping child always requires a diagnosis
- Knee pain may be referred to hip

REFERRAL

- Joint pain & fever
- Joint pain in an adolescent
- Abnormal hip examination
- Child refusing to move joint
- Limp & constitutional symptoms
- Limp in an overweight child
- Nocturnal bone pain
- Suspected malignancy (hepatosplenomegaly)

DEVELOPMENTAL DYSPLASIA OF THE HIP (DDH)

- M:F = 1:4
- Risk factors: breech delivery, first born, family history, oligohydramnios
- May not be detected in newborn period
- Presents with asymmetrical gluteal and thigh skin folds and Trendelenburg gait
- Positive Ortolani and Barlow signs in newborns
- Infants are treated with a Pavlik harness
- Children who present after 6 months or fail to respond may require surgery

SLIPPED UPPER FEMORAL EPIPHYSIS (SUFE)

- Most common hip disorder in adolescents
- Mean age at diagnosis is 13.5 years in boys and 12 years in girls, M>F
- Risk factors: obesity, endocrine disorders
- Bilateral in 25%
- Presents with limp, pain and inability to bear weight
- Orthopaedic emergency: further slippage may result in avascular necrosis
- Management is surgical

REFERENCES

- Tenore, Levine. *European Mastercourse in Paediatrics*. 2011.

DIFFERENTIAL DIAGNOSIS

COMMON CAUSES OF LIMPING IN CHILDREN

a. All ages

- Trauma (fracture, haemarthrosis, soft tissue)
- Infection (septic arthritis, osteomyelitis, discitis)
- Secondary to various viral illnesses
- Tumor
- Sickle cell disease
- Serum sickness

b. Toddler (1-3 years)

- Transient synovitis
- Toddler's fracture
- Child abuse (NAI)
- Developmental dysplasia of the hip
- Juvenile arthritis
- Neuromuscular disease
- Haemophilia
- Henoch-Schonlein purpura
- Rickets/Cerebral palsy

c. Child (4-10 years)

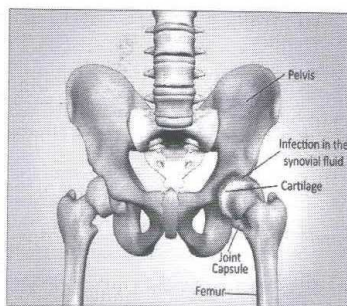
- Transient synovitis
- Juvenile arthritis
- *Perthes disease*
- *Rheumatic fever*
- Haemophilia
- Henoch-Schonlein purpura
- Kohler's Disease
- Rickets

d. Adolescent (11-16 years)

- Slipped upper femoral epiphysis
- Spondylosis
- Osgood-Schlatter
- Chondromalacia
- Overuse syndromes
- Osteochondritis dissecans

1. SEPTIC ARTHRITIS

- Can destroy a joint within 24 hours.
- Diagnosis is by exclusion.
- **Kocher's criteria for child with painful hip: "NEW T"**
 - Non-weight bearing on the affected side
 - ESR >40
 - WCC > 12
 - Temperature > 38.5
- Remember that not all of the features may be present and that the younger the child, the more subtle the presentation can be!
- **Investigations:**
 - **FBC, ESR, and CRP: negative results** do not rule the disease out.
 - **Blood cultures:** useful in identifying the organism but do not help confirm or exclude the diagnosis in the ED.
 - **X-ray:** used as useful baseline, can be initially normal.
 - **Lateral X-rays** may show bone destruction.
 - **Joint aspiration and synovial fluid analysis:** (most important diagnostic test): Fluid should be sent for **gram stain, cultures, crystal examination, and cell count.**
- **Management:**
 - IV Antibiotics: **Flucloxacillin and benzylpenicillin.**
 - **Analgesia:** consider splintage in addition to pharmacological treatment.
 - **Urgent orthopaedic referral:** for joint irrigation/drainage.



2. TRANSIENT SYNOVITIS

- It is a relatively common problem, especially in children between the ages of **3 and 6 years** old which is usually self-limiting within approximately one week.
- There is a higher incidence in boys than girls. Rapid onset of hip pain and limping in an otherwise well child. +/- history of **preceding viral illness**
- Hip held in flexion and abduction, limitation of internal rotation
- Only mild reduction of hip movements

3. PERTHE'S DISEASE

- *Avascular necrosis of the capital femoral epiphysis/osteonecrosis of the femoral head*
- Diagnosis is radiological but x-ray changes may be absent early in the illness.
- Age range **2-12 years** (majority **4-8yrs**)
- Boys: Girls = **4 :1**
- **15%** may be bilateral
- **Clinical:**
 - No systemic features
 - Present with pain and limp
 - Restricted hip motion on examination



*Perthe's disease of the left hip. 6-year-old girl with 1 week of pain in right hip. No history of trauma. There is **flattening and sclerosis of the left capital femoral epiphysis** in keeping with avascular necrosis, (red box). The right hip is of normal appearance.*

- **Imaging:**
 - The abnormalities are best seen on a **frog-leg lateral view**.
 - Perthe's disease **may be bilateral**, so it is important to look specifically for flattening and sclerosis of the femoral head & epiphyseal widening, not simply to compare the two sides. If the diagnosis is in doubt **MRI** is the investigation of choice.

4. SLIPPED UPPER FEMORAL EPIPHYSIS

- Late childhood/early adolescence, tends to occur in **10 – 15 year olds**
- Weight often > 90th centile.
- Boys: Girls = **2: 1**
- **25%** may be bilateral
- **Clinical:**
 - Presents with pain in hip or knee and associated limp.
 - The hip appears externally rotated and shortened.
 - There is decreased hip movement - especially internal rotation.
- **Risk Factors:** Obesity, Male sex, Immature skeleton, Family history of SUFE

- **Imaging:** AP views alone may miss subtle changes therefore **bilateral 'frog view'** is required



A 12-year-old girl with 2 weeks of pain in the left hip and knee, gradually worsening. Limping. Painful, restricted internal rotation.

The frogleg lateral shows a Type 1 (less than 33%) slippage of the capital femoral epiphysis ("melting ice cream cone"). A Klein line drawn through the centre of the femoral neck (red arrow) should bisect the capital epiphysis. It is clear from this that slippage has occurred. This condition can be bilateral so it is important to assess the position of the Klein line since the hips may appear symmetrical if both are abnormal. Diagnosis: **Left slipped upper femoral epiphysis**

5. OSTEOMYELITIS

- Osteomyelitis in children is usually secondary to haematogenous spread and therefore most commonly affects **the long bone metaphyses** as these are rapidly growing and highly vascular. It may spread to involve an adjacent joint.
- Symptoms are: **pain, redness, swelling & reduced use of the affected area** (which may be the only sign in infants). The diagnosis is easily missed in the early stages, so a high index of suspicion and close follow up is required.
- **Investigations:**
 - **FBC, ESR, CRP and Blood cultures.**
 - Microbiological diagnosis should be obtained from **blood, joint aspirate or bone aspirate** with Gram stain & culture.
 - **Bone biopsy** can be performed if other diagnoses are suspected, eg tumour.
 - **MRI** is the gold standard investigation to confirm the diagnosis.
 - **Plain radiographs** are initially normal but will show bone destruction once this develops, **usually 10-15 days** after the start of infection.



4-year-old with right knee pain; Non-weight-bearing & tender medial femoral condyle:

There is a **subtle ill-defined area of low density** approximately overlying the medial aspect of the metaphysis of the right distal femur (yellow box). This would be consistent with **osteomyelitis**.

6. AVULSION FRACTURES OF THE APOPHYSES

10-year-old boy, football injury, kicked ball and on swinging leg heard a pop in the right hip. Unable to weightbear, right leg externally rotated, dislocated or subluxed hip. There is a fracture of the right anterior inferior iliac spine (red box). This is due to avulsion by the **Rectus femoris muscle**.

- Avulsion fractures of the apophyses of the growing pelvis and hip are typically seen in **adolescent athletes (age range 14-25 years)** and are caused by forceful contraction of the muscle avulsing its bony attachment through the physis (growth plate).
- **Management**
 - Initially non-operative with **rest, ice, analgesia, passive and active mobilisation after 1 week**, and then a **gradual return to sporting activity**.
 - Occasionally delayed surgical fixation may be required for marked displacement & painful non-union.

7. PAEDIATRIC SARCOMAS

- Paediatric sarcomas are a heterogenous group of tumours that arise from primitive mesenchymal tissue.
- They can develop from smooth muscle, connective tissue, nerve or muscle at any site in the body and at any age from infancy onwards.
- Patients may present with a **painless mass** or with symptoms from adjacent tissues, eg pain, muscle weakness/reduced use of a limb, abnormal neurology, erythema, urinary retention, pathological fracture, back pain etc. They may also present with systemic symptoms such as **fever or weight loss**, particularly when metastases are present.
- Examination of a child with a suspected sarcoma must therefore be comprehensive, including:
 - Careful inspection and palpation of painful sites
 - Neurological examination for asymmetrical weakness or numbness
 - Respiratory examination for signs of metastases
 - Skin examination for purpura (thrombocytopaenia due to bone metastases).
- **ED investigation:**
 - **Plain x-ray:** Initially to look for soft tissue mass and signs of bone involvement such as erosion or pathological fracture.
 - **MRI:** is required to determine the extent of the disease, and should be done emergently if there is neurological involvement such as suspected spinal cord compression.



3-year-old girl with several months of left thigh pain, increasing, now swollen left anterior thigh and groin. There is **eccentric thickening** within the left proximal femur (red box). This would be consistent with an osteoid osteoma in appearance; however, there is also **a large soft tissue mass** at the proximal aspect of the left thigh (blue box). With the clinical information, this is extremely concerning for a sarcoma. (This was Ewing's sarcoma).

8. TODDLER'S FRACTURE

- A **toddler fracture** is a spiral break in the tibia (the shin bone in your leg), which occurs after a child twists their leg during a fall.
- It is a very low energy break, and usually it is a hairline crack without significant damage to the bone or surrounding tissue. *They are typically seen in children aged 9 months-3 years.*
- *Usually they are seen in the mid-shaft, but may occur in the upper or lower tibia*
- Toddler's fracture may present without specific history of trauma (although usually it does; 92% in one study)
- **Initial radiographs** will be normal (up to 29%) and so a presumptive diagnosis of toddler's fracture may be made in *an acute limp and no signs of infection*.
- *Radiographic evidence may only become apparent 7-10 days after the initial injury when new periosteal bone formation occurs.*
- There are no clinical features that can reliably differentiate toddler's fracture from other causes of limp.
- **ED ultrasound** examination of the limb may demonstrate **subperiosteal haematoma** in limping children with normal radiography.
- **Management of toddler's fracture should include:**
 - Consider NAI* as appropriate
 - Analgesia
 - Above knee back slab.
 - Follow up in **10-14 days in ED clinic** with another radiograph.
 - Weight bear as able
 - Advice to parents to return if condition worsens or if there are any ongoing concerns

*NAI: Non-Accidental Injury



9. OSGOOD-SCHLATTER DISEASE

- Osgood-Schlatter disease is a common cause of knee pain in growing adolescents.
- **It is an inflammation** of the area just below the knee where the patella tendon attaches to the tibial tuberosity
- **Risk factors for OSD include the following:**
 - Age: female 8-12 years & male between 12-15 years
 - Male sex (3:1)
 - Rapid skeletal growth
 - Repetitive sprinting and jumping sports
- Osgood Schlatter in adults can occur, especially if it has not been looked after during teenage years but is more unusual.
- **Activity** - As the young athlete's bones grow quickly, it can take some time for the muscles and tendons to catch up. These changes result in a pulling force from the patella tendon, on to the tibial tuberosity at the top of the shin. ***This area then becomes inflamed, painful and swollen.***
- This is frequent in younger people because their bones are still soft and are not yet fully grown. It is seen more often in children involved with **running and jumping activities** which put a much greater strain on the patella tendon.

**SYMPTOMS**

- Symptoms of Osgood Schlatters disease typically consist of pain at the **tibial tuberosity or bony bit at the top of the shin.**
- The tibial tuberosity may become swollen or inflamed and may even become more prominent than normal.
- Tenderness and pain is worse during and after exercise but usually improves with rest.
- The athlete is likely to experience pain when contracting the quadriceps muscles or performing squat type exercises.

TREATMENT

- **PRICE** principles
 - Protection of the knee from further injury.
 - Rest
 - Ice
 - Compression
 - Elevation

CHAPTER 18. RASHES IN CHILDREN

• INTRODUCTION

- o Children commonly present to the ED with a febrile illness and a rash.
- o Approximately 70% of these cases are attributable to viruses such as coxsackie, echovirus, and enteroviruses.
- o The rest result from bacterial infections such as Streptococcal and Staphylococcal infections (toxic shock syndrome), Mycoplasma, Rickettsial diseases, drug related causes or Kawasaki disease.

• Common prodromal symptoms include:

- o Irritability, Loss of appetite
- o Fever, Malaise
- o Headaches

- Originally six classical exanthems were described, however vaccination coverage has resulted in a fall in many of these illnesses and more recently newer exanthems such as **Gianotti Crosti Syndrome** have been described.

• Definitions

- o **An exanthem** is an eruptive skin rash associated with a fever or other constitutional symptoms. Exanthems may arise from an infectious disease or may be drug related.
- o **Enanthema** is an eruptive lesion on the mucous membranes occurring as a symptom of disease.

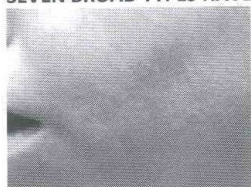
• HISTORY

o THE ORIGINAL SIX CLASSICAL CHILDHOOD EXANTHEMS

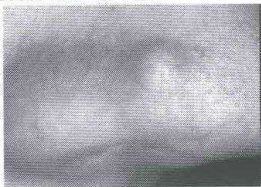
N ^o	OTHER NAMES	AETIOLOGY (IES)
1. First disease	Rubeola, Measles	Paramyxovirus
2. Second disease	Scarlet Fever or Scarlatina	Streptococcus
3. Third disease	Rubella, German measles, 3-day measles	Rubella virus
5. Fifth disease	Erythema infectiosum	Parvovirus B19
6. Sixth disease	Exanthem subitum, Roseola infantum,	Human Herpes Virus 6B Human Herpes Virus 7

CLINICAL ASSESSMENT

- Key points in history taking:
 - o Duration, Prodromal symptoms, associated symptoms such as itching, fever or pain, travel, unwell contacts, vaccination history, past medical history, drug allergies, recent medications.
- Examination should include records of:
 - o **Vital signs:** Pulse, Temperature, Capillary refill, Oxygen Saturations, RR
 - o **Distribution of rash:** Central or peripheral. Dermatomal distribution, extensor surfaces, mucosal involvement.
 - o **Appearance:** Rash colour, blanching or non-blanching, palpable, petechial
- Common rash descriptions
 - o Childhood exanthems vary greatly depending on factors such as location, size, elevation, palpability and the content of the associated skin eruptions.

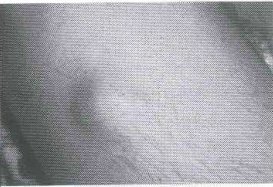
SEVEN BROAD TYPES HAVE BEEN IDENTIFIED:**Macule**

A macule is a circumscribed area of change in normal skin colour, with no skin elevation or depression. It may be any size.

**Papule**

A papule is a solid raised lesion up to 0.5 cm in greatest diameter.

Note, however, that some text definitions use 1.0 cm as a cut-off limit instead of 0.5 cm.

**Nodule**

A nodule is similar to a papule but is located deeper in the dermis or subcutaneous tissue.

Nodules are differentiated from papules by palpability and depth, rather than size

**Plaque**

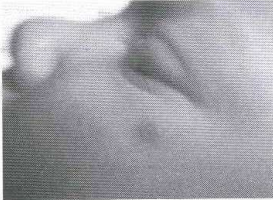
A plaque is an elevation of skin occupying a relatively large area in relation to its height.

It can often be formed by a confluence of papules

**Pustule**

A pustule is a circumscribed elevation of skin containing purulent fluid of variable character.

The fluid may be white, yellow, greenish or haemorrhagic.

**Vesicle**

A vesicle is a circumscribed, elevated, fluid-containing lesion less than 0.5 cm in its greatest diameter. It may be intra-epidermal or sub-epidermal in origin.

Note, however, that some text definitions use 1.0 cm as a cut-off limit instead of 0.5 cm.

Bulla

A bulla is similar to a vesicle, except the lesion is more than 0.5 cm in its greatest diameter.

Note, however, that some text definitions use 1.0 cm as a cut-off limit to replace 0.5 cm



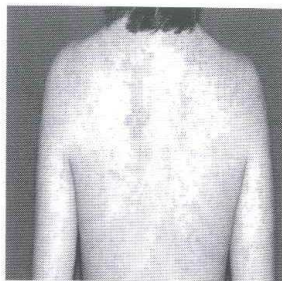
MACULOPAPULAR ERUPTIONS	DIFFUSE ERYTHEMA WITH DESQUAMATION	VESICOBULLOUS/ PUSTULAR ERUPTIONS
<ul style="list-style-type: none"> o Measles (rubeola) o Rubella o Erythema infectiosum (fifth disease) o Exanthum subitum (roseola) o Lyme disease o Drug related eruptions o Steven Johnsons Syndrome o Erythema Multiforme o Meningococcaemia 	<ul style="list-style-type: none"> o Scarlet fever o Toxic shock syndrome o Scalded skin syndrome o Kawasaki disease 	<ul style="list-style-type: none"> o (Diffuse) Varicella zoster o (Diffuse) disseminated Gonococcaemia o (Local) hand foot and mouth o (Local) Herpes zoster o Staphylococcal bacteraemia o Rickettsia

I. MEASLES

- This illness is caused by a **paramyxovirus** and is still a leading cause of morbidity and mortality in developing countries.
- Measles has been a notifiable illness since the 1940s.
- MMR vaccinations are administered at **12-15 months** of age and **3 to 5 years of age**.
- Side effects of the MMR vaccine are a **fever** usually in the second week post immunisation in 5 to 15 % of children and a rash.

Clinical assessment/ risks

- Measles occurs in epidemics in winter and spring, the infection is **spread by droplet spread or less commonly by aerosol spread**.
- The primary site of infection is the nasopharynx.
- Incubation period is approximately **7-21 days**.
- Infectivity is several days before the onset of the symptoms up to four days after the appearance of an erythematous maculopapular rash beginning on the head with a cephalocaudal progression.
- Individual lesions can coalesce and fade by the fifth day.
- *The clinical case definition of this disease has been defined as characterised by fever > 38.3°C or felt hot if not measured, a generalised maculopapular rash > 3 days and at least one of the 3 Cs "cough, conjunctivitis, coryza".*
- **Associated signs** described in measles are:
 - **Pin point elevations of the soft palate**, which coalesce to cause a reddened pharynx.
 - **Koplik spots**: These are blue white area sounded by erythema and occur on the buccal mucosa opposite the second molar. The last around 1-2 days and are pathognomonic of measles.
- **Complications:**
 - **ENT**: Otitis media, Tonsillitis, Laryngotracheobronchitis
 - **Respiratory**: Bronchopneumonia
 - **GIT**: Diarrhoea
 - **CNS**: Acute encephalitis, Sub acute sclerosing panencephalitis
- **Investigations strategies**
 - The investigation is performed using **oral fluid or serum sampling** for measles **IgM antibody**.
 - In acute cases measles can be detected using **throat swabs or in the urine**.



Koplik spots

MANAGEMENT OF MEASLES

- Treatment is largely symptomatic.
- **Prophylactic antibiotics** to children with measles in geographical areas with a high case fatality rate or with a high incidence of post-measles pneumonia. Antibiotics have been shown to reduce the incidence of complications of pneumonia, otitis media and tonsillitis.
- **Human Normal Immunoglobulin (HNIG)**
 - HNIG may be used to prevent or reduce the severity of an attack if used **within 72 hours** of contact in the following groups:

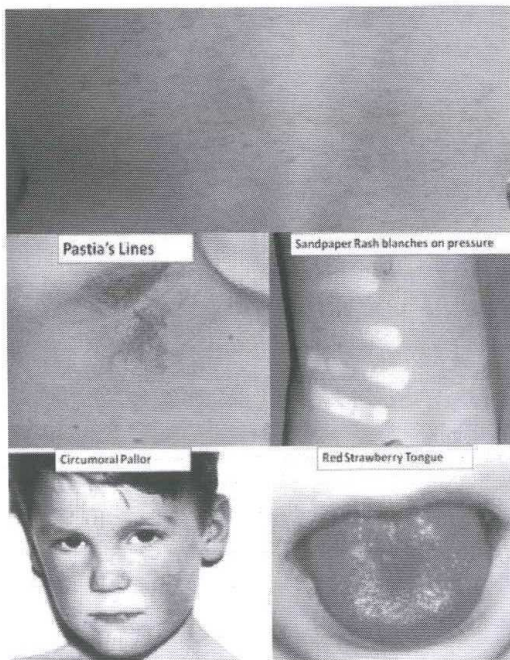
- Immunocompromised
 - Pregnant women
 - Infants under the age of 12 months
 - **MMR vaccine (13 months and 3.5 years)**
 - Is indicated in the healthy unimmunised or partially immunised within 72 hours of exposure to measles.
 - **Vitamin A therapy**
 - Vitamin A deficiency can be a risk factor for severe measles.
- Prognosis and follow up**
- **Notification:** this disease should be notified based on clinical suspicion.
 - Children should be kept off school until 5 days after the appearance of the rash.

II. SCARLET FEVER

- The organism is spread by **aerosol or droplet spread** and it can also be found in contaminated foods. The incubation period is usually **2-5 days**.
- The usual sites of infection are the **tonsils and pharynx**, although a broad spectrum of clinical presentations associated with this organism can occur.
- The risks of **post streptococcal glomerulonephritis** and **acute rheumatic fever** in the UK are low.

CLINICAL ASSESSMENT

- Scarlet fever is more common in childhood.
- The illness usually begins with a sore throat, headache, fever, tender cervical lymphadenopathy, malaise and also abdominal pain may occur in children.
- This is followed by a confluent **erythematous rash with a sandpaper like quality**.
- **Other features associated with Scarlet fever:**
 - **Pastias lines** in the flexural folds such as axilla, neck (due to linear petechiae formations.)
 - **Circumoral pallor**
 - **Pharyngitis**
 - **Desquamation of hands, feet and groin areas at 7-10 days**
 - **Red strawberry tongue**



- **COMPLICATIONS OF SCARLET FEVER**
 - **ENT:** Sinusitis, Mastoiditis, Peritonsillar Abscess
 - **Respiratory:** Pneumonia,

- **CVS:** Meningitis, Cerebral Abscess.
- **Systemic infections:** Septicaemia, Osteomyelitis, Septic Arthritis, Myocarditis and toxic shock like syndrome.
- **Renal:** Glomerulonephritis
- **Rheumatological:** Acute Rheumatic Fever.
- **INVESTIGATION STRATEGIES**
 - **Throat swab culture** (Gold standard test) can be performed, but a good quality specimen is required, results can take 24 to 48 hours to become available.
 - **Streptococcal antibody tests (ASOT)** are not indicated during acute illness
- **MANAGEMENT**
 - Antibiotic treatment is indicated (**Penicillin V or Erythromycin or Cephalosporin**) for **10 days**.
 - Notification
 - Patients diagnosed with this should have 5 days off from school or work following the commencement of antibiotics.

III. RUBELLA

- **DEFINITION**
 - Rubella is a self-limiting benign illness in adults and children. Spreads by **airborne transmission** or **droplet** spread from 7 days before to around 7 days after the onset of the rash. The incubation period is around **2 weeks** after which a prodrome of headaches, fever and lymphadenopathy occur.
 - Immunisation programmes have had a major impact on this illness in developed countries and the incidence of rubella has been markedly reduced in the UK
- **CLINICAL ASSESSMENT**
 - Rubella is associated with a characteristic macular rash, which starts on the face, passing down through the body to the feet and is associated with a fever, tender occipital and posterior auricular lymphadenopathy, arthralgia and respiratory involvement.
 - **Forscheimer spots:** are pin point red macules and petechiae, which may be seen on the soft palate and uvula during the rash phase.
 - The clinical signs of Rubella may be difficult to distinguish from other viral illnesses such as Parvovirus B19, measles, dengue, Human herpes virus 6. Therefore, laboratory diagnosis is required.
 - This vital in especially during pregnancy due to consequences to the foetus
- **COMPLICATIONS**
 - Encephalitis, hepatitis, pericarditis,
 - Neuritis
 - Conjunctivitis
 - Orchitis
 - Arthralgia /Arthritis is more common in postmenopausal women.
 - Haemolytic anaemia, Thrombocytopenia



Forscheimer spots

CONGENITAL RUBELLA SYNDROME

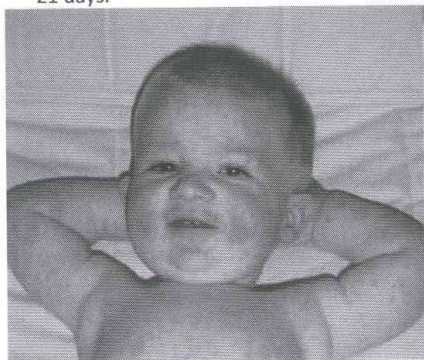
- The occurrence of congenital defects is the highest (85%) if the mother develops the rash in the first 12 weeks of pregnancy.
- **COMPLICATIONS:**
 - **Ophthalmological:**
 - *Cataracts, Microphthalmos and Congenital glaucoma*
 - **Cardiac:**
 - *Patent ductus arteriosus (PDA) and Peripheral pulmonary artery stenosis*
 - **Auditory:**
 - *Sensory neural hearing impairment*
 - **Neurological:**
 - *Meningoencephalitis*
- **INVESTIGATION STRATEGIES**
 - **IgG and IgM assays are used.**
 - The clinical diagnosis of rubella is unreliable and therefore laboratory confirmation and follow up of this disease is important.
 - This is vital especially during pregnancy due to the consequences to the foetus.

MANAGEMENT

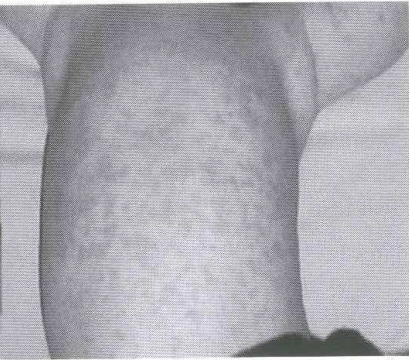
- Children or adults with Rubella should remain of school for at **least 5 days** after the onset of the rash.
- This is a notifiable disease.
- Women should avoid pregnancy until 3 months after immunisation.
- **MMR vaccine (13 months and 3.5 years)**

IV. PARVOVIRUS B19

- **Erythema infectiosum** is caused by **Parvovirus B19**.
- This Virus is a highly infectious human pathogen found globally.
- Illness arising from this virus can present with a wide spectrum of clinical features.
- The virus is predominately spread in **respiratory droplets** but can also pass from **mother to foetus** and in **blood transfusions**.
- The incubation period of this illness is usually between **4 and 14 days** but can be as long as 21 days.



Stage 1: Slapped cheek appearance



Stage 2: maculopapular rash to the trunk and limbs

CLINICAL ASSESSMENT

- o Fever and nonspecific symptoms occur early and this is followed approximately 2 to 3 weeks later by a rash and arthropathy.
- o The classical rash (**stage 1**) has been described as a **slapped cheek appearance**, which lasts for up to 4 days. The rash is a confluent erythematous, oedematous rash with patches or plaques on cheeks with sparing of nasal bridge and periorbital areas
- o This is followed by (**stage 2**) a **maculopapular rash to the trunk and limbs**. This rash can vary in intensity and duration. As the rash begins to fade it can take on a lacy appearance. Arthropathy can occur in around 5% of the paediatric population and up to 60% of the adult population.
- o Children tend to follow a milder course whereas in adults a symmetrical arthropathy affecting hands, wrists and feet can be more severe.
- **Clinical spectrum** of illness associated with Parvovirus B19:
 - o Arthropathy, Henoch schonlein purpura, Autoimmune disorders
 - o Myocarditis, Hepatitis, Papular purpuric glove and socks syndrome
 - o Meningitis and encephalitis
 - o Fibromyalgia and chronic fatigue syndrome
 - o Chronic infection in patients with immunodeficiency

COMPLICATIONS

- Complications of parvovirus infections are seen in the following groups:
 - o Haemoglobinopathies: **aplastic crises**
 - o Immunocompromised
 - o Pregnant women: **hydrops fetalis**
- Transient aplastic crises can occur in those with and without underlying chronic haemolytic illness.
- Approximately 60% of women are immune to this virus. Viral transmission in pregnancy is more likely to occur during the first and second trimester.
- **Foetal hydrops** is more likely to occur in the second trimester.

INVESTIGATION STRATEGIES

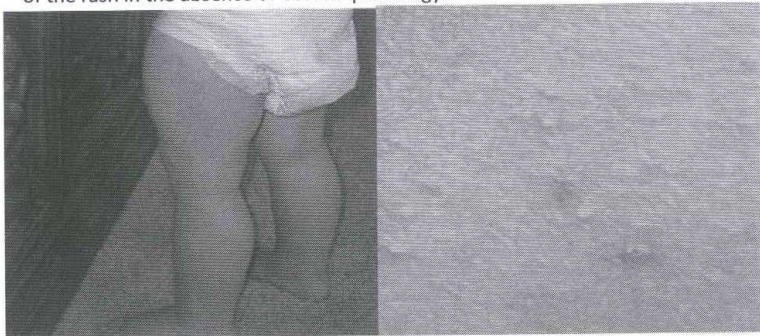
- o **IgM antibodies** appear around 10 days' post infection and remain detectable for up to 2-3 months.
- o **IgG antibodies** appear at about 14 days' post infection and remain for life.
- o Pregnant women in contact with Parvovirus B19 or with suspected parvovirus B19 should have serological testing and referral to the obstetricians for regular monitoring and follow up.

MANAGEMENT

- o For the majority of patients Erythema infectiosum is **self-limiting**.
- o **Analgesia** may be needed for joint pains.
- o **Transfusion** may be indicated for patients with aplastic crises.
- o **Intravenous immunoglobulin**, which contains pooled neutralising anti B19 antibody has been used to treat immunocompromised patients
- o Children in high risk groups should be **referred**.

V. ROSEOLA INFANTUM/ EXANTHEM SUBITUM

- Human Herpes virus 6 (HHV-6) or HHV-7 causes **Exanthem subitum** (sixth disease or **Roseola infantum**). An HHV6 infection results in a common benign illness in children aged 6 months to 2 years old and is a common cause of fever and febrile seizures in the infant.
- The rash of Roseola infantum can be confused with Measles and Rubella.
- HHV 6 is very wide spread and nearly all children will acquire the infection early in childhood. The incubation period is from **5 to 15 days**.
- **CLINICAL ASSESSMENT**
 - The illness is associated with a mild respiratory illness, 3-5 days of a high fever of 39-40°C and cervical lymphadenopathy (30%).
 - The fever disappears coinciding with a rash. Around 10% of US infants are reported to develop the characteristic rash **commencing behind the ears i.e. discrete blanching macules and papules which are surrounded by halos**.
 - The rash lasts around 1-2 days. Palpebral edema has been observed before the onset of the rash in the absence of ocular pathology.



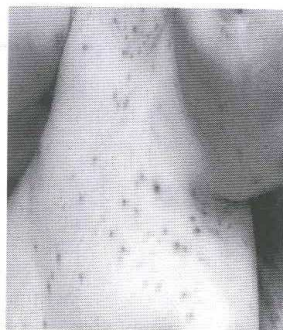
- **SYMPTOMS AND SIGNS SEEN IN EXANTHEM SUBITUM**
 - Palpebral oedema in 30%
 - Uvulopalatal junction ulcers.
 - Erythematous papules on the soft palate also known as **Nagayamas spots** (65%)
 - Diarrhoea (68%)
 - Cough (50%)
 - Prenatal and perinatal infections are uncommon due to maternal antibodies.
- **INVESTIGATION STRATEGIES**
 - For the majority of cases serological testing is not necessary.
- **COMPLICATIONS ASSOCIATED WITH EXANTHEM SUBITUM:**
 - Meningoencephalitis
 - Encephalitis
 - Hemiplegia
- **MANAGEMENT**
 - HHV6 causes a benign illness and in the majority of cases antiviral therapy is not needed.
 - Reactivation of this virus can occur in transplant recipients.
- The rash is often misdiagnosed as measles.

VI. CHICKEN POX (VARICELLA ZOSTER)

- Chicken pox (varicella) is a common world wide highly infectious illness and primarily a disease of childhood. The illness is spread via respiratory droplets.
- Incubation is from **10 to 21 days**.
- The period of infectivity is from the time when symptoms first appear until all lesions have crusted over. This is usually around 5-6 days after the onset of the illness. Most crusts will disappear by 20 days.
- **Symptoms:** This is acoryzal type illness with **itchy fluid filled vesicles**, which progress over the trunk around 3-5 days.
- It is possible to be infected with no symptoms. Fever tends to resolve by day 4.
- *Prolonged fever > 4 days should prompt the suspicion of complications of Varicella such as secondary bacterial sepsis.*

• COMPLICATIONS

- Most children have a mild illness with no complications.
- The risks to the mother are the highest in the third trimester and the risks to the foetus are the greatest in the first and second trimester.
- Groups which may be at a risk of greater severity of illness with chicken pox:
 - Neonates
 - Immunocompromised
 - Pregnant women
 - Patients with chronic steroid use



• COMPLICATIONS OF CHICKEN POX

- *Pneumonia*
- *Bacterial superinfection of skin*
- *Bacteraemia/ toxic shock syndrome*
- *Encephalitis*
- *Acute cerebellar ataxia*
- *Necrotising fasciitis*
- *Purpura fulminans/ disseminated zoster*
- *Thrombocytopenia*
- *Glomerulonephritis*
- *Arthritis/ Hepatitis*



• CONGENITAL COMPLICATIONS

- *Shortened limbs*
- *Skin scarring*
- *Cataracts*
- *Growth retardation.*
- There is currently no routine immunisation against Varicella in the UK.
- Since 2003 vaccination is recommended to non-immune health care workers.



• MANAGEMENT

- **Oral acyclovir** reduces the total number of lesions and the duration of fever when used within 24 hours of the onset of rash in immunocompetent children.
- It has not been shown to reduce the incidence of VZV pneumonia or complications when compared to placebo. The results do not support the widespread use of acyclovir in the immunocompetent child.
- **Varicella zoster immunoglobulin** should be given to neonates whose mothers develop the rash 7 days before or 7 days after the delivery, to reduce the risk of severe neonatal Varicella.
- Neonates presenting with a chickenpox rash **should be admitted for acyclovir 10mg/Kg.**
- In the majority of cases simple treatment advice can be given and the child can be managed at home.
- Children should be kept off school until all the lesions have crusted over and no new crops have occurred, this is usually **around 5-6 days.**
- Patients with chickenpox should **avoid contact** with pregnant women, neonates and the immunocompromised.

VARICELLA ZOSTER AND PREGNANCY

- Any pregnant woman who has not had chickenpox or who is found to be seronegative for **VZV IgG** should be advised to minimize any contact with chickenpox and shingles and to seek medical help immediately if exposed. If a pregnant woman is exposed, the first course of action is to perform a blood test and check for **VZV immunity.**
- If she is not immune and the history of the exposure is significant, she should be given **VZV immunoglobulin** as soon as possible. It is effective **up to 10 days after being exposed.** A pregnant woman that develops chickenpox should seek medical help urgently. There is an increased maternal risk of **Pneumonia, Encephalitis** and **Hepatitis** as well as the 1% risk of developing **FVS.**
- **Acyclovir** should be used with caution before 20 weeks gestation, but is recommended after 20 weeks if the woman presents within 24 hours of the onset of the rash.

SUMMARY OF CHILDHOOD EXANTHEMS

Diseases	Etiology	Description
Measles	Paramyxovirus	An erythematous maculopapular rash beginning on the head
Scarlet fever	Group A beta-haemolytic streptococci	A confluent erythematous rash with a sandpaper-like quality
Rubella	Rubella virus	Macular in form, starting on the face then passing down the body to the feet
Erythema infectiosum	Parvovirus B19	Resembling a slapped cheek and lasting up to 4 days
Exanthem subitum	Human herpesvirus 6 or Human herpesvirus 7	Discrete blanching macules and papules surrounded by halos
Chicken pox	Varicella virus	Fluid-filled vesicles which progress over the trunk
Gianotti-Crosti syndrome	A range of different viruses and bacteria	Papular or papulovesicular in form and distributed symmetrically on the face, buttocks and extremities

VII. KAWASAKI'S DISEASE

• BACKGROUND

- Kawasaki's is a disease of exclusion and the diagnosis and treatment of possible cases must be discussed with senior medical staff.

• DIAGNOSIS

- There is no diagnostic test and diagnosis is based on clinical criteria and the exclusion of other diseases. Infection must be considered and often in practice children are treated with antibiotics for 24-48 hours.
- The criteria may present sequentially such that an 'incomplete' case can evolve with time to become 'complete'.
- This makes the definite exclusion of Kawasaki's difficult and **the disease should be considered in any irritable child with a fever for 5 or more days.**

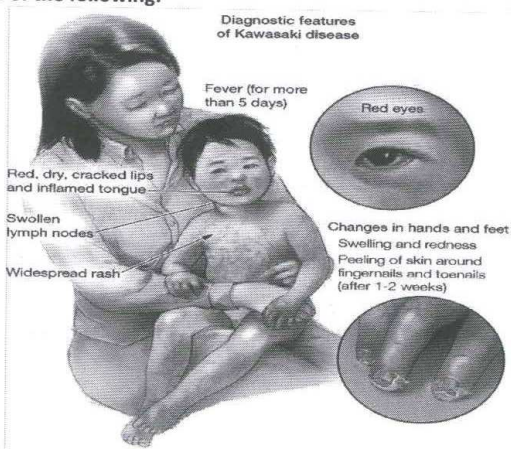
• DIAGNOSTIC CRITERIA

- **Fever more than 5 days plus 4 of the following:**

- Conjunctivitis
- Lymphadenopathy
- Rash
- Changes to lips or oral mucosal (strawberry tongue)
- Changes of extremities

- **Differential Diagnosis**

- Toxic Shock Syndrome
- Scalded skin syndrome
- Scarlet fever
- EBV, CMV, Mycoplasma
- Polyarteritis nodosa
- Juvenile idiopathic arthritis
- Malignancy (lymphoma)



• INITIAL INVESTIGATIONS

- K.D. is associated with many non-specific laboratory findings.
 - Acute phase proteins raised
 - Neutrophilia, ESR raised
 - Thrombocytosis towards the end of the second week and therefore is not useful diagnostically

- LFTs may be deranged
- Pyuria, CSF pleocytosis

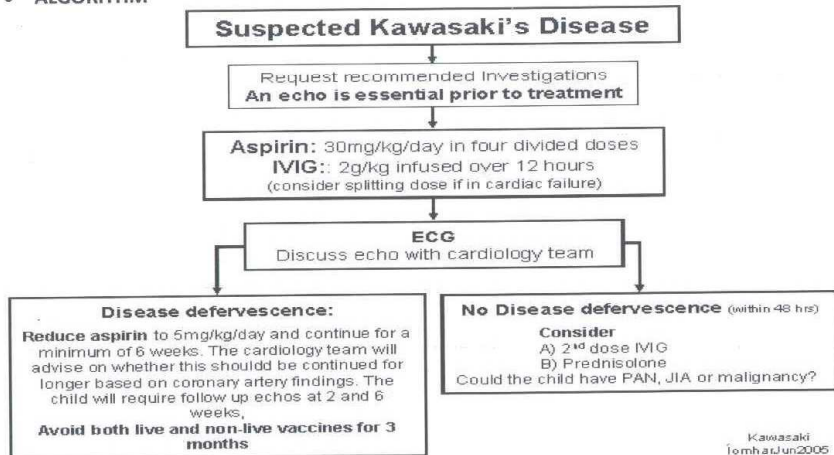
○ OTHER INVESTIGATIONS:

- FBC and Film, ESR, CRP, Renal profile, LFT, Coagulation
- Autoimmune profile
- ASOT, anti-DNAse Serology: mycoplasma, enterovirus, adenovirus, measles, parvovirus, EBV, CMV
- Blood Cultures, Urine MC&S
- ECG and echocardiogram/ Consider CXR

- TREATMENT**

- o **Aspirin:** Given during the acute phase of the illness at high dose (**30mg/kg/day**) and then reduced to **5mg/kg/day** when the inflammatory markers have returned to normal.
- o **Immunoglobulin:** Early recognition and treatment with IVIG has been shown to reduce the occurrence of coronary artery aneurysms. For maximum benefit it should be given before day 10 of the illness but should not be withheld if diagnosed after this time. If you suspect KD then it should be treated regardless of what the echo shows. Recommended dose is **2g/Kg over 12 hours** except where there is cardiac compromise when a smaller volume in divided doses may be preferable.

- ALGORITHM**



VIII. PEMPHIGOID

- Background**

- o Commonest autoimmune sub-epidermal blistering disorder.
- o Auto-antibodies target "adhesion complexes" in the skin's basement membrane=blister formation.
- o High relapse rate.

- Clinical**

- o Itchy⁺⁺⁺, tense fluid filled blisters skin and/or mucous membranes.
- o Usually limbs, groin, and abdomen.
- o Older patients.
- o Beware septicaemia (especially if immunocompromised).

- Differential Dx**

- o Bullous pemphigoid.
- o Linear IgA disease.
- o Epidermolysis bullosa acquisita.
- o Bullous drug reaction.



INVESTIGATIONS

- o Clinical diagnosis.
- o Confirmatory biopsy +/- immunofluorescence.
- o ↑CRP & ↑ESR.

MANAGEMENT

- o **Potent topical steroids** (clobetasol propionate).
- o **Oral steroids or Dapsone** (esp. for mucous membrane disease).
- o **Tetracyclines +/- nicotinamide** in milder cases.
- o **Immunosuppressants** (MTX / Azathioprine) in severe cases.
- o **Regular skin antiseptic soaks.** And protective non-adherent dressings.

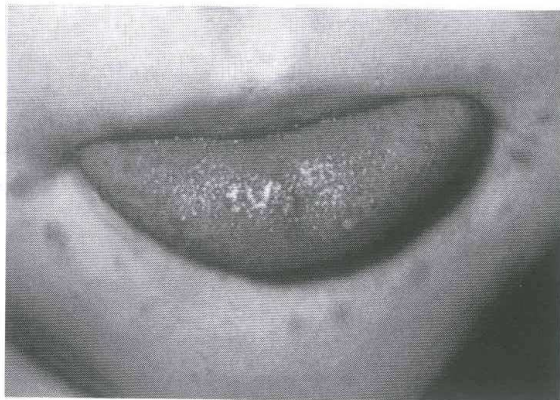
IX. HAND, FOOT AND MOUTH DISEASE

CLINICAL

- o **Coxsackie virus** (more serious Enterovirus clusters in Asia)
- o Person to person spread.
- o Max children **1 - 4 years** (common up to 10 years).
- o Sores in mouth, rashes on hands & feet and buttocks.
- o "Textbook" vesicles occur at junction of hard and soft skin (palm, sole/ankle area).
- o Commonest cause of mouth sores (painful, small yellow sores, red halo) children.
- o Painful eating then hand rash.
- o May have low fever, anorexia, sore throat, abdominal pain for 7 days.
- o Usually mild but beware dehydration and occasional arthropathy.

CLINICAL DIAGNOSIS

- o Rarely progress to encephalitis / transverse myelitis (flaccid - so do neuro exam).
- o Hydration, analgesia (mouth ulcers), control pyrexia.
- o Keep from school only if ill (may attend school if rash alone).



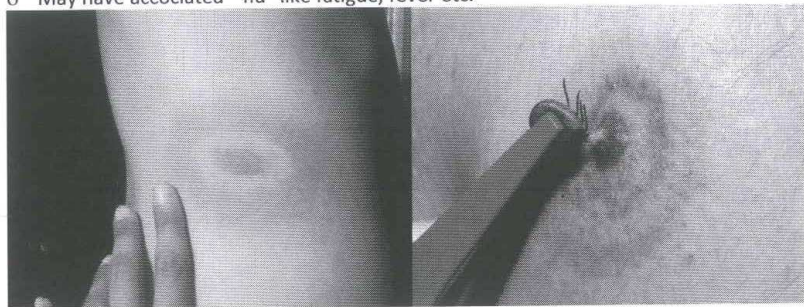
X. LYME DISEASE

- **BACKGROUND**

- **Spirochete *Borrelia burgdorferi* infection via tick bite**

- **CLINICAL**

- **Erythem migrans** - macular erythematous rash with central clearing
 - Starts at bite site (3-21 days later) and spreads (spirochetes migrate from wound site)
 - Rash disappears after 1 month or may never be noticed
 - May have associated "flu" like fatigue, fever etc.



- **Late symptoms seen in 2/3 if untreated:**

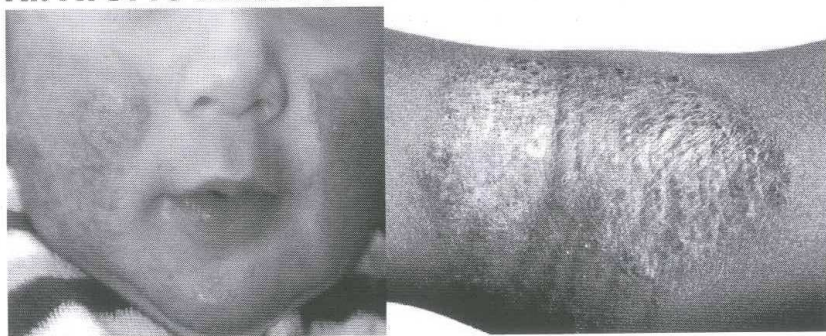
- **Neuroborreliosis:** Mononeuritis Multiplex (e.g. Bell's) or Meningitis
 - **Oligoarthritis:** (knees) - Synovial ↑WCC and PCR + Positive for spirochetes
 - **Pancarditis:** AV Block, or Myocarditis

- **INVESTIGATIONS**

- **Serology testing** to confirm suspicious cases.

- **MANAGEMENT**

- If erythema migrans or strong suspicion for Lyme disease - treat with **Doxycycline**, then ask for serology testing.
 - If neuroborreliosis - admit neurology
 - If oligoarthritis get (sterile procedure) synovial aspirate
 - If cardiac, beware heart block (atropine ± pacing) and admit cardiology
 - Please contact microbiology for antibiotic advice

XI. ATOPIC ECZEMA

INTRODUCTION

- o Atopic eczema (atopic dermatitis) is a chronic inflammatory skin condition.
- o It is common in childhood affecting about 10% of school-aged children.
- o Moderately severe eczema is miserable for the whole family.
- o Itch and sleep deprivation are the main complaints.
- o Topical treatment is messy and time consuming.
- o Many parents who end up bringing their child to the ED will be exhausted and fed up.
- o It is important that they get consistent, clear messages about treatment.
- o Flares are common, and sometimes there will be a treatable exacerbating factor such as infection.

ACUTE MANAGEMENT

- One of the commonest reasons for acute flare up is secondary infection.
- This is almost always with **staphylococcus aureus**.
- **BACTERIAL INFECTION**
 - o Crusted weepy areas suggest bacterial infection with *S. aureus*.
 - o Take bacterial swabs to confirm sensitivities.
 - o Treat with **oral flucloxacillin for 10 days or erythromycin if penicillin allergic**.
 - o Frankly infected eczema should not be bandaged – wait 48-72 hours into antibiotic.
 - o Treatment before starting bandaging.
 - o **Fusidic acid** containing creams (Fucibet, Fucidin) should be limited to short term use (ie 5 days for localised infection) because of bacterial resistance.
- **VIRAL INFECTION**
 - o **Herpes simplex** causing 'eczema herpeticum' with monomorphic punched out erosions and vesicles.
 - o Infection can rapidly become widespread and cause severe systemic upset.
 - o Take viral fluid for slides and swabs to confirm diagnosis.
 - o Oral treatment with **aciclovir** if infection localised and no systemic upset.
 - o IV treatment if there is widespread infection or systemic upset: senior review is indicated.
 - o Take viral samples



- **LONG TERM MANAGEMENT:**

- o Staying with simple treatments that you know well and spending time explaining and encouraging correct usage is often more effective than using yet another different preparation.

- **SOAP SUBSTITUTES**

- o Soap and shampoo must be avoided.
- o Effective cleansing can usually be achieved by applying the child's usual emollient prior to washing and then rinsing it off. Alternatively bath and shower oils may be preferred.
- o **Bath:** Oilatum bath additive 500mls or Balneum bath additive 500mls
- o **Shower:** Dermol 500

- **EMOLLIENT (MOISTURISER)**

- o Emollients are the mainstay of treatment. It is really important to continue using these to keep the skin in good condition even when the eczema is quiet.
- o The best emollient is the one the patient will use adequate amounts of.
- o Greasy emollients are often more effective and need to be applied less often, but are sticky and mark clothing and some patients/ parents will find them unacceptable.
- o They should be applied generously at least twice a day, and more frequently to drier areas. Children should use about **250g of emollient/ week**.
- o Adolescents should use about **500g of emollient/week**.

- **TOPICAL STEROIDS**

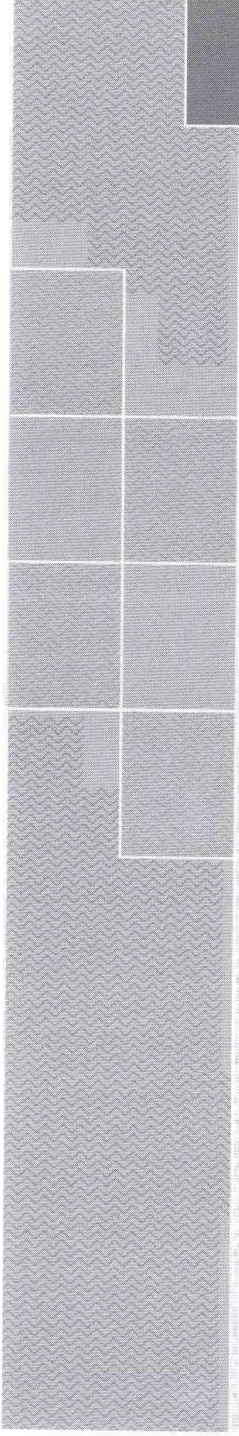
- o Steroids are safe if used appropriately. Parents are often wary of steroids and it is common to use too little, too late
- o Enough steroid should be applied to make the skin appear slightly shiny
- o Almost all preparations come in a cream or ointment form, which needs to be specified when prescribing. Cream is water based and therefore will mix with wet, weepy areas. Ointments are greasy and better for dry areas.
- o Just changing the "vehicle" may make the same strength of steroid work better.
- o A suitable steroid regimen for moderate eczema would be:
 - **1% hydrocortisone ointment/ cream** (weak steroid) applied twice daily to red/eczematous areas on face and neck supplied in 30g tube
 - **Eumovate ointment/cream** (moderately potent steroid) applied twice daily to red/eczematous areas on trunk and limbs supplied in 100g tube

- **BANDAGES**

- o Bandages are a useful adjunct to treatment. They improve the penetration of topical treatments into the skin, feel soothing, provide a barrier to scratching and prevent emollient making clothes greasy. Bandages can be used over night or continuously changing the bandages once or twice in a 24-hour period:
 - Dry – Tubular bandage
 - Wet- wet wrapping technique puts damp layer of bandages under dry layer
 - Paste- impregnated (sticky) bandages under tubular bandages- "Viscopaste" (zinc oxide) or "Ichthopaste" (zinc oxide and ichthammol)

- **ANTIHISTAMINICS**

- o Antihistamines may be useful at night for sedating effect: **Piriton, Vallergan, Phenergan**
- o Use a decent, sedative dose at bedtime. The child may get tolerant of the sedative effect, so intermittent use when most needed makes sense.



PRACTICAL PROCEDURES

12 QUESTIONS

**Compiled & Edited by:
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CHAPTER 1. ARTERIAL CANNULATION

• IMPORTANT LANDMARK FOR ARTERIAL CANNULATION

- **The radial artery:** volar wrist, medial and proximal to the radial styloid.
- **The femoral artery:** between the symphysis pubis and anterior superior iliac spine. lateral to the femoral vein, medial to the femoral nerve.
- **The brachial artery:** in the medial antecubital fossa, lateral to the medial epicondyle, medial to the biceps brachii tendon.
- **Dorsalis pedis artery:** highly variable; locate with palpation of the pulse; in the midfoot, lateral and parallel to the extensor hallucis longus tendon.

• INDICATIONS

- Serial Arterial Blood Gas monitoring
- Frequent blood sampling
- Continuous Blood Pressure monitoring

• CONTRAINDICATIONS

- **Absolute:**
 - Absent pulse
 - Raynaud syndrome / Buerger disease
 - Full-thickness burns over the cannulation site
 - Inadequate circulation to the extremity
- **Relative:**
 - Anticoagulation & Coagulopathy
 - Atherosclerosis
 - Inadequate collateral flow
 - Infection at the cannulation site
 - Partial-thickness burn at the cannulation site
 - Previous surgery in the area
 - Synthetic vascular graft

• COMPLICATIONS

- Arterial Thrombosis
- Occult bleeding
- Cerebral embolization
- Localized infection

• TECHNIQUE: PREPARATION FOR ARTERIAL CANNULATION

- Perform **Allen Test** to confirm collateral circulation
- Heparinize syringe
 - Start with 10 cc Syringe with stopcock
 - Draw up 3-5 ml of Heparinized saline (50 units/ml)
- Obtain IV catheter
 - Needle of 18 or 20 gauge with plastic cannula
 - Flush with Heparinized saline
- Position patient's wrist and hand
 - Patient dorsiflexes wrist over towel pad
 - Tape palm and upper Forearm to arm board
- Clean radial entry site
 - Povidone-Iodine solution (betadine) scrub and Alcohol scrub
- Local anesthetic at entry site
 - Small skin wheal (1-2 ml) of Lidocaine 2%

CHAPTER 2: PERIPHERAL VENOUS CANNULATION**• INDICATIONS**

- Administration of intravenous medicines.
- Transfusions of blood or blood components.
- Maintenance or correction of hydration levels if unable to tolerate oral fluids.
- Potential venous access.

• CONTRAINDICATIONS

- Extremities that have massive edema,
- Burns or injury
- The presence of infection as suggested by inflammation, phlebitis, cellulitis.
- The presence of injury or damage (e.g. fracture, Stroke, oedema, lymphadenopathy).
- Veins which are mobile or tortuous, or sited near a bony prominence.
- If intravenous therapy is predicted to be long-term.
- Continuous infusions or therapies which are vesicant or have a pH of 9.

• COMPLICATIONS

- Thrombophlebitis
- Leakage

• PERIPHERAL IV SITES

- The preferred site in the emergency department is the **veins of the forearm**, followed by the **median cubital vein** that crosses the antecubital fossa.
- In trauma patients, it is common to go directly to the **median cubital vein** as the first choice because it will accommodate a large bore IV and it is generally easy to catheterize. In circumstances where the veins of the upper extremities are inaccessible, the veins of **the dorsum of the foot** or **the saphenous vein** of the lower leg can be used. In circumstances in which no peripheral IV access is possible a central IV can be started.

1. VENOUS CUTDOWN**• INDICATIONS**

- Saphenous vein cutdown is indication for the purpose of **emergency venous access** when attempts to gain access via peripheral or percutaneous routes have failed.
- Burns, Shock, Asystole or PEA, Sclerosed veins of IVDU

• CONTRAINDICATIONS

- Alternative option exists for venous access
- Coagulopathy or bleeding diathesis
- Vein thrombosis
- Overlying cellulitis
- Major trauma at the proposed site
- Injury proximal to the proposed site

• COMPLICATIONS

- Failed cannulation
- Creation of a false passageway in the vessel wall
- Hemorrhage
- Air embolus
- Venous thrombosis
- Infection
- Nerve / Artery/ Vein transection
- Damage of surrounding structures

- **LANDMARK**
 - Locate the **great saphenous vein** 1 cm anterior and 1 cm superior to the medial malleolus
- **PATIENT PREPARATION**
 - Local anesthesia is used (1% lidocaine with or without epinephrine).
 - The patient is placed in a supine position with the foot externally rotated.
 - A tourniquet can be placed above the ankle but is not necessary.

2. INTRAOSSEOUS CANNULATION

- **INDICATIONS**
 - Urgent venous access is required after **3 failed attempts** at venous cannulation
 - **Difficulty in establishing venous access**, as in the following settings: Burns, Obesity, Edema, Seizures
 - **Condition necessitating rapid high-volume fluid infusion**, such as the following: Hypovolemic shock, Burns
 - **Afford access to the systemic venous circulation**, as with the following: Cardiopulmonary arrest, Burns, Blood draws, Local anesthesia and Medication infusion
- **LANDMARKS**
 - If possible, avoid areas of burns or of skin infection
 - **Proximal tibia:** Anteromedial surface, 2-3 cm below the tibial tuberosity
 - **Distal tibia:** Proximal to the medial malleolus
 - **Distal femur:** Midline, 2-3 cm above the external condyle
 - Sternum, Deltoid, Iliac crest

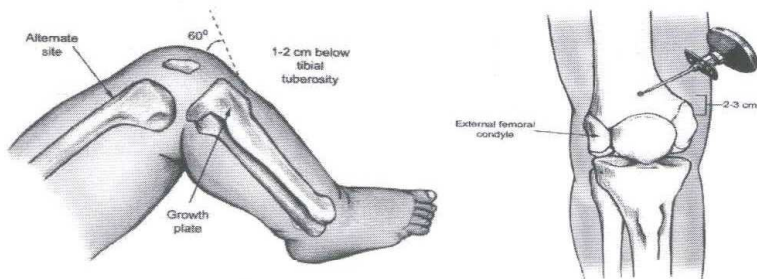


Figure 6.2.1. Landmarks of IO cannulation

- **CONTRAINDICATIONS**
 - Proximal ipsilateral fracture
 - Ipsilateral vascular injury
 - Osteogenesis imperfecta
 - Osteoporosis
- **COMPLICATIONS**
 - Failure to enter the bone marrow, with extravasation or subperiosteal infusion
 - Through and through penetration of the bone
 - Osteomyelitis (rare in short term use)
 - Growth plate injury
 - Local infection, skin necrosis, Pain
 - Compartment syndrome, fat and bone microemboli have all been reported (rare)

CHAPTER 3. CENTRAL VENOUS CANNULATION**• INDICATIONS FOR CENTRAL VENOUS CATHETERISATION**

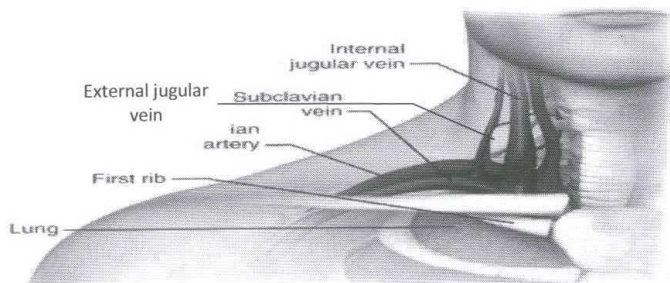
- **Access for drugs**
 - Infusion of irritant drugs—for example, chemotherapy
 - Total parenteral nutrition
 - Poor peripheral access
 - Long term administration of drugs, such as antibiotics
- **Access for extracorporeal blood circuits**
 - Renal replacement therapy
 - Plasma exchange
- **Monitoring or interventions**
 - Central venous pressure
 - Central venous blood oxygen saturation
 - Pulmonary artery pressure
 - Temporary transvenous pacing
 - Targeted temperature management
 - Repeated blood sampling

• CONTRAINDICATIONS TO CENTRAL VENOUS CATHETERISATION

- Coagulopathy
- Thrombocytopenia
- Ipsilateral Haemothorax or Pneumothorax
- Vessel thrombosis, Stenosis, or Disruption
- Infection overlying insertion site
- Ipsilateral indwelling central vascular devices

• COMPLICATIONS OF CENTRAL VENOUS CATHETERIZATION

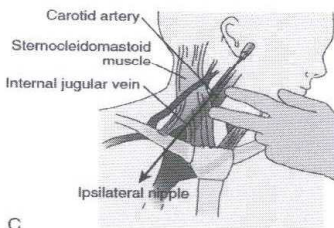
IMMEDIATE	DELAYED
<ul style="list-style-type: none"> ▪ Bleeding ▪ Pneumothorax or Hemothorax ▪ Arterial puncture ▪ Arrhythmia ▪ Air embolism ▪ Thoracic duct injury (with left SC or left IJ approach) ▪ Catheter malposition 	<ul style="list-style-type: none"> ▪ Infection ▪ Venous thrombosis ▪ Pulmonary emboli ▪ Catheter migration ▪ Catheter embolization ▪ Myocardial perforation ▪ Nerve injury



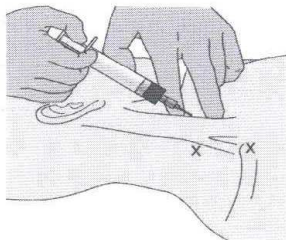
1. INTERNAL JUGULAR APPROACH

- **Anatomy:** from jugular foramen \rightarrow joins subclavian vein behind sternal extremity of clavicle
- **Central Approach:** find 1cm above the apex of head of SCM and clavicle \rightarrow 60 degrees to skin aiming towards ipsilateral nipple (blood should be obtained within 3cm)
- **Lateral/Posterior Approach:** find 2-3 finger breaths above clavicle along posterior border of SCM, direct needle towards jugular notch (blood should be aspirated within 5cm)
- **Anterior Approach:** identify the **carotid artery** and **mid point of medial SCM border**, aim toward ipsilateral nipple.

ANTERIOR APPROACH



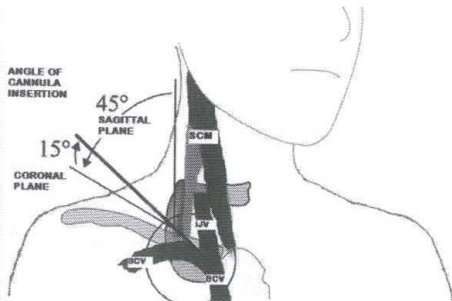
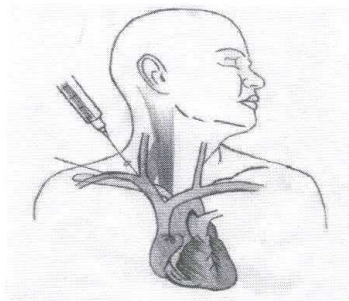
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2. SUBCLAVIAN APPROACH

- **Approaches:** Supraclavicular, Infraclavicular and Lateral
- The objective of the **supraclavicular technique** is to puncture the subclavian vein in its superior aspect just as it joins the internal jugular vein.
 - Identify the **clavisternomastoid angle** formed by the junction of the lateral head of the sternocleidomastoid muscle and the clavicle.
 - Raise the patient's head to make this landmark more apparent.
 - *The needle is inserted 1 cm lateral to the lateral head of the sternocleidomastoid muscle and 1 cm posterior to the clavicle and directed at a 45-degree angle to the sagittal and transverse planes and 15 degrees below the coronal plane aiming toward the contralateral nipple.* The needle bisects the clavisternomastoid angle as it is advanced in an avascular plane, away from the subclavian artery and the dome of the pleura, entering the junction of the subclavian and internal jugular veins.
 - **The right side** is preferred because of the lower pleural dome, more direct route to the SVC, and absence of thoracic duct.



3. FEMORAL APPROACH

- **Anatomy:**
 - NAVEL (nerve – artery – empty space – lymph node (lateral to medial))
 - Boundaries of the femoral triangle are adductor longus and sartorius
- **Approach:** slight external rotation of hip, palpate pulse, medial to arterial pulsation

• **ADVANTAGES AND DISADVANTAGES OF CENTRAL VEIN APPROACHES**

APPROACH	ADVANTAGES	DISADVANTAGES
External jugular	<ul style="list-style-type: none"> • Superficial vessel that is often visible • Coagulopathy not prohibitive • Minimal risk of pneumothorax (especially with US guidance) • Head-of-table access • Prominent in elderly patients • Rapid venous access 	<ul style="list-style-type: none"> • Not ideal for prolonged venous access • Poor landmarks in obese patients • High rate of malposition • Catheter may be difficult to thread
Internal jugular	<ul style="list-style-type: none"> • Minimal risk of pneumothorax (especially with US guidance) • Head-of-table access • Procedure-related bleeding amenable to direct pressure • Lower failure rate with novice operator • Excellent target using US guidance 	<ul style="list-style-type: none"> • Uncomfortable • Not ideal for prolonged access • Risk of carotid artery puncture • Thoracic duct injury possible on left • Dressings and catheter difficult to maintain • Poor landmarks in obese/edematous patients • Potential access and maintenance issues with concomitant tracheostomy • Vein prone to collapse with hypovolemia • Difficult access during emergencies when airway control being established
Subclavian	<ul style="list-style-type: none"> • Easier to maintain dressings • More comfortable for patient • Better landmarks in obese patients • Accessible when airway control is being established 	<ul style="list-style-type: none"> • Increased risk of pneumothorax • Procedure-related bleeding less amenable to direct pressure • Decreased success rate with inexperience • Longer path from skin to vessel • Catheter malposition more common (especially right SCV) • Interference with chest compressions
Femoral	<ul style="list-style-type: none"> • Rapid access with high success rate • Does not interfere with CPR • Does not interfere with intubation • No risk of pneumothorax • Trendelenburg position not necessary during insertion 	<ul style="list-style-type: none"> • Delayed circulation of drugs during CPR • Prevents patient mobilization • Difficult to keep site sterile • Difficult for PA catheter insertion • Increased risk of iliofemoral thrombosis

CHAPTER 4. ARTERIAL BLOOD GAS SAMPLING

• INDICATIONS FOR ABG SAMPLING

- To interpret oxygenation levels
- To assess for potential respiratory derangements
- To assess for potential metabolic derangements
- To monitor acid-base status
- To assess carboxyhaemoglobin in CO poisoning
- To assess lactate
- To gain preliminary results for electrolytes and Haemoglobin
- Can be conducted as a one-off sample or repeated sampling to determine response to interventions

• CONTRAINDICATIONS TO ABG SAMPLING

- **Absolute:**
 - Absent pulse
 - Thromboangiitis obliterans (Buerger disease)
 - Full-thickness burns over the cannulation site
 - Inadequate circulation to the extremity
 - Raynaud syndrome
- **Relative:**
 - Anticoagulation/ Coagulopathy
 - Atherosclerosis
 - Inadequate collateral flow
 - Infection at the cannulation site
 - Partial-thickness burn at the cannulation site
 - Previous surgery in the area
 - Synthetic vascular graft

• EQUIPMENT REQUIRED FOR ABG SAMPLING

- Gloves, sharps bin, Cleaning swab
- Gauze, Tape, ABG syringe

• PROCEDURE FOR ARTERIAL BLOOD GAS (ABG) SAMPLING

- Consent the patient verbally after explaining the procedure
- Set up a tray with a sharps bin
- Expel excess heparin from ABG syringe
- Palpate for radial pulse
- Transfix artery between forefinger and middle finger
- Insert ABG syringe into palpated artery
- Depending on the syringe it may self fill or you may need to withdraw the plunger carefully. Remove needle and syringe after sample gained (only 1-2ml required)

• POST PROCEDURE CARE

- Apply pressure to area with gauze and tape.
- Advise patient to continue giving pressure for 5-10 minutes
- Take sample to the analyser as soon as possible
- Ensure the result is labelled with the patient's details and documented in the notes
- Ensure inspired oxygen concentration is clearly documented
- Ensure adequate pressure on sampled area to prevent a haematoma

• IN THE EVENT OF FAILURE

- Call for senior help

CHAPTER 5. LUMBAR PUNCTURE

• INDICATIONS

- Suspicion of Meningitis
- Suspicion of Subarachnoid Hemorrhage (SAH)
- Suspicion of central nervous system (CNS) diseases such as Guillain-Barré syndrome and Carcinomatous Meningitis.
- Therapeutic relief of Pseudotumor Cerebri

• CONTRAINDICATIONS

- Possible raised intracranial pressure
- Thrombocytopenia or other bleeding diathesis
- Suspected spinal epidural abscess

• COMPLICATIONS

- Post-LP headache
- Infection
- Bleeding
- Back pain
- Cerebral herniation
- Minor neurologic symptoms such as radicular pain or numbness

• Indications for performing brain CT scanning before lumbar puncture in patients with suspected meningitis include the following:

- *Altered mental Status*
- *Focal neurologic signs*
- *Papilloedema*
- *Seizure within the previous week*
- *Patients who are immunocompromised*
- *Patients who are older than 60 years*
- *Patients with known CNS lesions*

• LANDMARK

- **The posterior iliac crests** are easily palpated in most patients. A line drawn between the **superior border of the posterior iliac crests will intersect the L4 spinous process.**
- Using this surface landmark, **the L3-L4-L5 interspaces can be localized.**
- Identify the **L4-L5 interspinous process space** midline as the needle insertion site.
- If insertion at this space is unsuccessful, try the **L3-L4 space.**
- To avoid damaging the spinal cord, do not go above the **L2- L3 space. The L4-L5 interspace is likely the largest when the spine is flexed, and therefore this space should be attempted first.**

Layers: Lumbar puncture: "SSS I LED AS"

- Skin
- Superficial fascia
- Supraspinous ligament
- Interspinous ligament
- Ligamentum flavum
- Epidural space
- Dura mater
- Arachnoid
- Subarachnoid space containing cerebrospinal fluid

CHAPTER 6. PLEURAL TAP AND ASPIRATION

• BEDSIDE ULTRASONOGRAPHY

- Before the procedure, **bedside ultrasonography** can be used to determine the presence and size of pleural effusions and to look for loculations.
- During the procedure, **bedside ultrasonography** can be used in real time to facilitate anesthesia and then guide needle placement.

• INDICATIONS

- Symptomatic treatment of large pleural effusions or for treatment of empyemas.
- Pleural effusions of any size that require diagnostic analysis.

• CONTRAINDICATIONS

- Uncorrected bleeding diathesis
- Chest wall cellulitis at the site of puncture

• COMPLICATIONS

- Pneumothorax, Hemothorax, Lung laceration, Infection, Empyema
- Damage to the intercostals or internal mammary vessels,
- Diaphragmatic injury,
- Puncture of the liver or spleen,
- Damage to other abdominal organs,
- Abdominal hemorrhage,
- Reexpansion pulmonary edema,
- Air embolism,
- Cough & Pain
- Risk of Catheter fragment left in the pleural space.

• PERIPROCEDURAL CARE

- **Informed Consent:** should be obtained from the patient or parent if minor.
- Provide a focused set of risks and complications.
- Discuss how these risks can be avoided or prevented (eg, proper positioning, ensuring that the patient remains as still as possible during the procedure, adequate analgesia).

• PATIENT PREPARATION

- Patient preparation includes **adequate anesthesia** and **proper positioning**.

• ANESTHESIA

- In addition to **local anesthesia**, **mild sedation** may also be considered.
- **IV Midazolam or Lorazepam.**
- The skin, subcutaneous tissue, rib periosteum, intercostal muscle, and parietal pleura should all be well infiltrated with local anesthetic.

• POSITIONING

- **Patients who are alert and cooperative** are most comfortable in a **seated position**, leaning slightly forward and resting the head on the arms or hands or on a pillow, which is placed on an adjustable bedside table. This position facilitates access to the posterior axillary space, which is the most dependent part of the thorax.
- **Unstable patients** and those who are unable to sit up **may be supine** for the procedure.

• LANDMARK

- Traditionally, this is **between the 7th and 9th rib spaces** and between the **posterior axillary line and the midline**.

• **DIAGNOSTIC ANALYSIS OF PLEURAL FLUID**

- The following laboratory tests should be requested:
 - *pH level*
 - *Gram stain*
 - *Culture*
 - *Cell count and Differential*
 - *Glucose level, protein levels, and lactic acid dehydrogenase (LDH) level*
 - *Cytology*
 - *Creatinine level* if Urinothorax is suspected
 - *Amylase level* if esophageal perforation or pancreatitis is suspected
 - *Triglyceride levels* if chylothorax is suspected (eg, after coronary artery bypass graft [CABG], especially if the inferior mesenteric artery [IMA] was used; milky appearance is not sensitive)

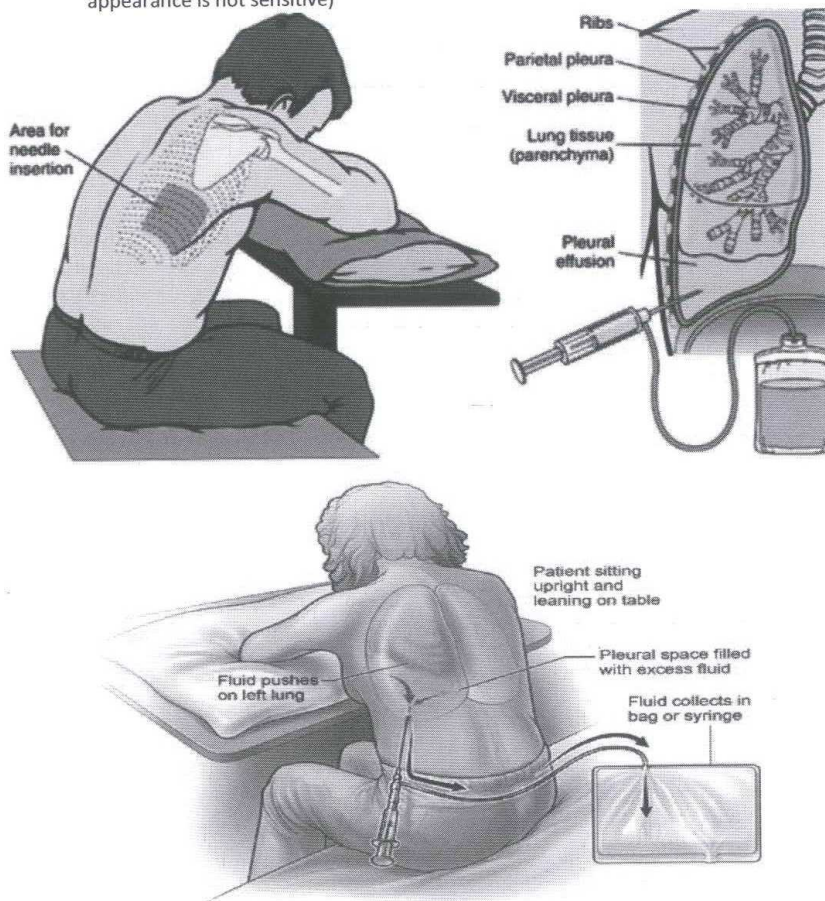


Figure 6.6.1. Landmarks of Pleural tap aspiration

CHAPTER 7. INTERCOSTAL DRAIN-OPEN

- **INDICATIONS FOR INTERCOSTAL DRAIN INSERTION (CHEST DRAIN/PLEURAL DRAIN)**
 - **Absolute Indications**
 - Pneumothorax (Tension, Open or Simple)
 - Haemothorax
 - Pleural effusion
 - Empyema and Chylothorax
 - To facilitate Pleurodesis
 - **Relative Indications**
 - Rib fractures & Positive pressure ventilation
 - Profound hypoxia / hypotension & penetrating chest injury
 - Profound hypoxia / hypotension and unilateral signs to a hemothorax
- **CONTRAINDICATIONS**
 - Coagulopathy
 - Local infection
- **COMPLICATIONS**
 - Pain, Bleeding, Infection.
 - Damage to local structures,
 - Incorrect placement (extrapleural, in the fissure, drainage holes outside the pleura, tube kinked)
 - Pulmonary injury and bronchopleural fistula.
 - Insertion into a vascular structure (pulmonary artery or left ventricle).
 - Tube blockage, displacement and dislodgement, Infection.
 - Misuse of drainage system leading to introduction of air or fluid into pleural cavity.
 - Recurrence of underlying condition
 - Wound dehiscence and Scarring
- **PRE-PROCEDURE**
 - **Written consent:** should be gained for Pain, failure of procedure, bleeding, infection, damage to surrounding structures and pneumothorax if the procedure is for an effusion.
 - **Aseptic technique:** All drains should be inserted with full aseptic precautions (washed hands, gloves, gown, antiseptic preparation for the insertion site and adequate sterile field) in order to avoid wound site infection or secondary empyema.
 - **Patient position:** The patient should be positioned appropriately; this will depend on the reason for insertion and the clinical state of the patient. The most commonly used position is with the patient lying at 45° with their arm raised behind the head to expose the axillary area or in a forward lean position. The procedure may also be performed with the patient lying on their side with the affected side uppermost. In trauma situations emergency drain insertion is more likely to be performed whilst the patient is still in supine as part of the primary trauma survey.
 - **Premedication/local anaesthetic**

LANDMARKS

- **The 5th intercostal space anterior to the mid-axillary line** for most situations.
- This area is commonly known as the **"safe triangle"**.
- Any other placement should be discussed with a senior clinician (apical pneumothorax), placement of a chest tube in the **2nd intercostal space** should be considered.

- A specific position may also be required for a loculated effusion.

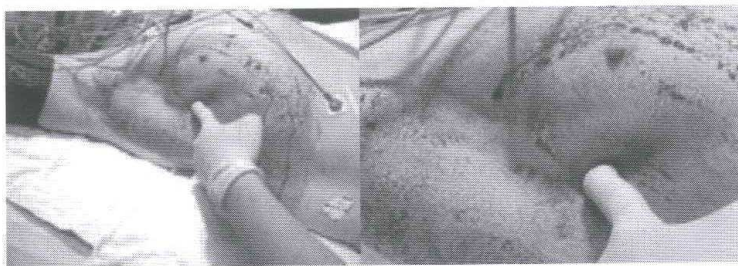


Figure 6.7.1. Open ICD insertion

TRIANGLE OF SAFETY

- The triangle of safety is an anatomical region in the **axilla** that forms a guide as to the safe position for intercostal catheter (ICC) placement.
- With the arm abducted, the apex is the axilla, and the triangle is formed by the:
 - Lateral border of the **pectoralis major** anteriorly
 - Anterior border of the **latissimus dorsi** posteriorly
 - Inferiorly, by a line superior to the horizontal level of the nipple and an apex below the axilla

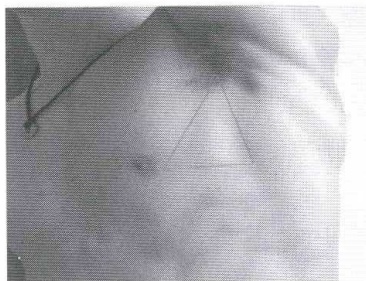
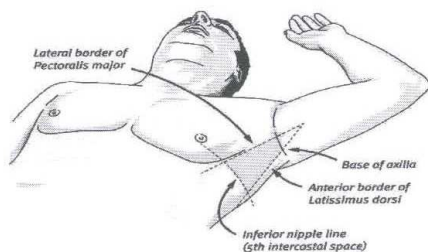


Figure 6.7.2. Safe triangle for chest drain insertion

○ INSERTION

- Full asepsis
- 2-3cm transverse incision on top of rib
- Blunt dissection down to pleura (just superior to rib -> avoid neurovascular structures)
- End point: pleural cavity (hiss or blood)
- Sweep with finger, insert clamped drain using curved forceps to guide in
- Connect to UWSD, check for drainage and respiratory swing
- Suture, Sterile dressing

○ POST INSERTION

- CXR
- Watch for complications:
 - **Not draining:** check for kinking
 - **Organ injury** (lung, liver, spleen, heart, vessel): careful insertion
 - **Blood loss:** careful observation
 - **Surgical emphysema:** small hole and good suturing
 - **Infection:** sterile technique

CHAPTER 8. INTERCOSTAL DRAIN - SELDINGER

• INDICATIONS OF SELDINGER TECHNIQUES

- Angiography,
- Insertion of chest drains and central venous catheters,
- Insertion of PEG tubes using the push technique,
- Insertion of the leads for an artificial pacemaker or implantable cardioverter-defibrillator, and
- Numerous other interventional medical procedures.

• COMPLICATIONS

- Hemorrhage: Puncture of the intercostal artery.
- Organ perforation.
- Infection: non-aseptic technique.
- Inadequate "stay" suture allowing the chest tube to fall out.
- Tube blockage
- Pneumothorax if the procedure is for an effusion.

PROCEDURE

- **Consent the patient** for pain, failure of procedure, bleeding, infection, damage to surrounding structures and pneumothorax if the procedure is for an effusion.
- **Small-bore Seldinger chest drain insertion**
 - Ensure full aseptic conditions are maintained at all times
 - Perform surgical hand wash, don apron and sterile gloves
 - Open pack keeping contents sterile, take additional equipment from assistant in a sterile fashion
 - Clean the skin using Antiseptic skin preparation such as Chlorhexidine
 - Apply sterile drape
 - Make a small incision (3-5 mm) in the skin where the drain is to be inserted
 - Using the needle and syringe in the pack gently insert (avoiding excess force) towards the upper border of the chosen rib aspirating continuously until air in the syringe confirms the position of the needle in the pleural cavity
- **Best Practice Statement**
 - *Air or fluid must be aspirated before wire is inserted (stop and get help or arrange USS guidance if you cannot achieve this). Both the needle and dilator should be inserted without force*
- **Rationale:** Confirm correct position and minimise risk of damage to underlying structures
 - Hold the needle steady and remove the syringe. Feed the wire gently through the needle into the pleural cavity (**AT ANY STAGE IN THE PROCEDURE ONE HAND SHOULD ALWAYS BE HOLDING THE WIRE**)
 - Remove the needle leaving the guide-wire in place, make sure that the wire does not shear. Feed the first dilator down over the wire and into the pleural cavity. It may be necessary to make the incision a few mm bigger. Repeat the process for the second dilator if present. Note: the dilator only needs to be inserted to a depth that is sufficient for the chest wall to be dilated.
 - Over insertion of the dilator risks damage to thoracic structures and has been identified by NPSA as a significant cause of morbidity with the Seldinger technique.
 - Remove the dilator leaving the wire in place. Estimate the depth of insertion on the scale on the drain from the apex to the skin.

- Feed the 12F chest drain over the wire until it is in the pleural cavity to desired depth.
- Remove the wire making sure that the chest drain stays in position **DO NOT LET GO OF THE DRAIN NOW.**
- Attach the end of the drain onto the underwater seal system and make sure that the chest drain bottle is placed below the patient.
- Check that the water in the chest drain is bubbling or swinging, if in doubt ask the patient to cough gently
- An adhesive dry dressing such as MEPORE is normally all that is required to secure the drain to the skin
- Remove the drape
- Dispose of all waste and sharps appropriately

- **POST-PROCEDURE**

- Place drain on free drainage but monitor closely
 - If the patient has a chronically collapsed lung and you drain more than 1-1.5l in the first 24 hours there is risk of **re-expansion pulmonary oedema**
- **Analgesia**
- **Post procedure CXR**
- **Document procedure** clearly and document length of drain inserted
- **Advise patient** to always hold drain bottle below level of insertion
- **Respiratory review** and advise on onward management

- **IN THE EVENT OF FAILURE**

- Stop procedure
- Seek senior help
- Re-review imaging and patient with a senior colleague to ensure presence of fluid
- Consider further imaging or chest drain insertion in radiology

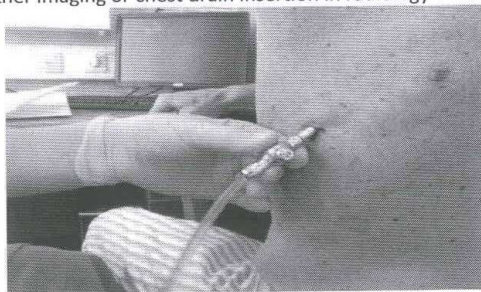


Figure 6.8.1. Seldinger Intercostal drain

CHAPTER 9. ASCITIC TAP (PARACENTESIS)

- **Abdominal paracentesis** is a simple bedside or clinic procedure in which a needle is inserted into the peritoneal cavity and ascitic fluid is removed.
- **Diagnostic paracentesis** refers to the removal of a small quantity of fluid for testing.
- **Causes of transudative ascites include the following:**
 - Heart failure
 - Hepatic cirrhosis
 - Alcoholic hepatitis
 - Fulminant hepatic failure
 - Portal vein thrombosis
- **Causes of exudative ascites include the following:**
 - Peritoneal carcinomatosis
 - Inflammation of the pancreas or biliary system
 - Nephrotic syndrome
 - Peritonitis
 - Ischemic or obstructed bowel
- **INDICATIONS**
 - **Diagnostic tap** is used for the following:
 - New-onset ascites - Fluid evaluation helps to determine etiology, differentiate transudate versus exudate, detect the presence of cancerous cells, or address other considerations.
 - Suspected spontaneous or secondary bacterial peritonitis
 - **Therapeutic tap** is used for the following:
 - Respiratory compromise secondary to ascites
 - Abdominal pain or pressure secondary to ascites (including abdominal compartment syndrome)
- **CONTRAINDICATIONS**
 - An acute abdomen that requires surgery
 - Severe thrombocytopenia
 - Coagulopathy
 - Pregnancy
 - Distended urinary bladder
 - Abdominal wall cellulitis
 - Distended bowel
 - Intra-abdominal adhesions
- **COMPLICATIONS**
 - Failed attempt to collect peritoneal fluid
 - Persistent leak from the puncture site
 - Wound infection
 - Abdominal wall hematoma
 - Spontaneous hemoperitoneum - This rare complication is due to mesenteric variceal bleeding after removal of a large amount of ascitic fluid (>4 L).
 - Hollow viscus perforation (small or large bowel, stomach, bladder)
 - Catheter laceration and loss in abdominal cavity
 - Laceration of major blood vessel (aorta, mesenteric artery, iliac artery)

- Postparacentesis hypotension
- Dilutional **hyponatremia**
- Hepatorenal syndrome

EQUIPMENT REQUIRED FOR ASCITIC TAP (PARACENTESIS)

- Ultrasound (ideally)
- Dressing trolley & sharps bin.
- Sterile field. Sterile dressing pack. Sterile gloves. ...
- Analgesia. 10mls of 1% or 2% Lidocaine. Orange (25G) needle (x1) ...
- 20ml Syringe (x1) with green (19G) needle (x1)
- Specimen containers.
- Blood culture bottles.
- Dressing.

PRE-PROCEDURE

- **Consent patient and explain procedure:** *Consent for infection, bleeding, pain, failure, damage to surrounding structures (especially bowel perforation – rare), leakage*
- **Positioning:** *Lie patient flat and examine clinically to confirm ascites*
- **Ultrasound** area for insertion
- **Define landmarks:** *Aim for 1/3 to 1/2 of the way between the anterior superior iliac spine and the umbilicus avoiding vessels and scars.*

PROCEDURE FOR ASCITIC TAP (PARACENTESIS)

- Position the patient supine in the bed with their head resting on a pillow.
- Select an appropriate point on the abdominal wall in the right or left lower quadrant, lateral to the rectus sheath. If a suitable site cannot be found with palpation and percussion consider using ultrasound to mark a spot.
- Clean the site and surrounding area with 2% Chlorhexidine and apply a sterile drape.
- Anaesthetise the skin with Lidocaine using the orange needle.
- Anaesthetise deeper tissues using the green needle, aspirating as you insert the needle to ensure you are not in a vessel before infiltrating with lidocaine. Use a maximum of 10mls of Lidocaine.
- Take a clean green needle and 20ml syringe and insert through the skin advancing and aspirating until fluid is withdrawn
- Aspirate 20ml Remove needle and apply sterile dressing

TECHNICAL CONSIDERATIONS

- Depending on the clinical situation, fluid may be sent for the following laboratory tests:
 - **Gram stain**
 - **Cell count** (elevated counts may suggest infection)
 - **Bacterial culture**
 - **Total protein level**
 - **Triglyceride levels** (elevated in chylous ascites)
 - **Bilirubin level** (may be elevated in bowel perforation)
 - **Glucose level**
 - **Albumin level**, used in conjunction with serum albumin levels obtained the same day (used to calculate SAAG; see the Ascites Albumin Gradient calculator)
 - **Amylase level** (elevation suggests pancreatic source)
 - **Lactate dehydrogenase (LDH) level**
 - **Cytology**

CHAPTER 10. AIRWAY PROTECTION

I. AIRWAY ASSESSMENT

1. STRATEGY

- **Testing for gag reflex**
 - It is part of the American Academy of Neurology's criteria for determining brain death.
 - The gag reflex is one of the body's airway protective mechanisms and is easily and commonly tested. Its absence or presence is used as a guide to determine if either an oral or nasopharyngeal airway should be placed during CPR.
- **Evaluate the patient's ability to swallow.**
 - When a patient gags, the muscles of their soft palate and pharynx contract collectively but briefly. The stimulus for this is relayed by the ninth and tenth cranial nerves.
 - Patients who have secretions pooling in the back of their mouths demonstrate some degree of swallowing dysfunction and may be at much greater risk for aspiration than those who do not have a gag reflex.

2. BASIC AIRWAY ASSESSMENT

- **Patency** is our primary concern in airway assessment, but the patient who can talk does not necessarily have a **PATENT** airway.
- Think about a patient with bad airway edema from a laryngeal fracture, or a patient with an inhalational injury. They can talk, but their airway may be closing nonetheless.
- The ability to talk usually implies a **PROTECTED** airway, not a **patent** airway.
- Lack of airway protection is a less urgent matter than patency.
- **PATENCY** is assessed through the presence/absence of obstructive symptoms (stridor, secretions, snoring, etc.), or findings suggesting an airway that may become obstructed (singled nasal/facial hair, carbonaceous sputum, stab to neck with risk of expanding hematoma).

3. MANAGING THE AIRWAY

- The next issue is that the indications for definitive airway management **involve both airway and breathing**, so just because the airway is patent and protected, it doesn't mean the patient won't need to be intubated.
- In general, the **indications for intubation** are (Walls et al., 2012):
 - *Failure to oxygenate*
 - *Failure to ventilate*
 - *Failure to maintain airway patency*
- Look before you Leap: Assess the airway for difficulty of potential interventions
- From here, there is confusion about assessing the airway for **PATHOLOGY** (patency and protection issues) versus **INTERVENTIONS** such as:
 - *Bag-Valve Mask ventilation (MOANS),*
 - *Difficult Laryngoscopy & Endotracheal Intubation (LEMON),*
 - *Difficult Laryngeal Mask Airway (RODS) and*
 - *Difficult Surgical Airway (SHORT).*
- *Deciding that something is wrong with the airway SHOULD be the easy part, and simple interventions will deal with the most immediate airway issue of obstruction. (These simple interventions being: suction, jaw thrust, oral-pharyngeal airway, supplemental O2).*

- Deciding if a *definitive* intervention (i.e. intubation) is required and how to go about that takes a lot more experience and training.
- The table below contains mnemonics for assessing a patient for intubation, bag-valve-mask ventilation, LMA placement, and cricothyrotomy.

AIRWAY MNEMONICS MODIFIED FROM THE STARS™

- *Manual & Walls et al. (4th edition)*

Difficulty Endotracheal Intubation	Difficult Bag-Mask-Valve (BMV)
<ul style="list-style-type: none"> • L Look externally • E Evaluate 3-3-2 • M Mallampati • O Obstruction/Obesity • N Neck Mobility 	<ul style="list-style-type: none"> • M Mask seal (Beard, Blood...) • O Obstructed/Obese • A Age > 55 • N No teeth/Neck Stiffness / Neck Mass • S Stridor / Snore/ Stiff Lungs
Difficult Laryngeal Mask Airway (LMA)	Difficult Cricothyrotomy
<ul style="list-style-type: none"> • R Restricted Mouth Opening • O Obstruction • D Distorted airway anatomy • S Stiff Lungs / Neck 	<ul style="list-style-type: none"> • S Surgery • H Hematoma, Have Infection (Abscess) • O Obesity • R Radiation • T Trauma, Tumor

4. APPROACH TO BASIC AIRWAY ASSESSMENT

- The approach to airway assessment for PATHOLOGY is to assess the “S’s” in 3 steps:

THE S’S OF AIRWAY PATHOLOGY

- **Step 1: Is there evidence of airway OBSTRUCTION now – is it complete or partial?**
 - **Complete Obstruction:**
 - Silence without chest rise or
 - See-Saw Chest movement (chest down, abdo up with attempted respiration – resulting from diaphragmatic excursion with a closed glottis/obstructed tongue)
 - *Complete airway obstruction needs **immediate intervention** – cardiac arrest is likely within seconds to minutes of complete airway obstruction.*
 - **Partial Obstruction**
 - Stridor – airway swelling/compression by hematoma
 - Secretions – saliva, blood
 - Snoring – tongue relaxation
 - Smash – risk of teeth/blood in the airway
 - **Interventions:**
 - **SIMPLE** = jaw thrust, suction, OPA, supplemental Oxygen, BVM
 - **ADVANCED** = Definitive airway management
- **Step 2: Is there a risk of ANTICIPATED airway obstruction?**
 - Singe or Sputum (carbonaceous) – risk of delayed airway swelling from inhalational burn
 - Stab or Swelling neck – risk of delayed airway compression from expanding hematoma or neck mass
 - **Interventions:**
 - Frequent re-assessment,
 - Early intervention (if skilled),
 - Early consultation with experts.

- **Step 3: Is there a risk of Aspiration from failure to PROTECT their airway?**
 - Sleepy (low GCS)
 - **Interventions:**
 - Definitive airway management if:
 - Decreased LOC (impaired gag reflex) is going to be prolonged, is not easily reversible, is deteriorating, or
 - If intubation will facilitate further investigations (ie CT Scan).
 - The level of consciousness can always change and along with it the ability to protect the airway from aspiration, however aspiration is not an immediate event the second your GCS hits 8 or less.
 - **GCS < 8 is not an absolute indication for intubation.** Many intoxicated patients and post-ictal patients live with a GCS of 5 or 6 and wake up a few hours later.
 - They don't usually aspirate, and can be managed without definitive airway protection.
 - Alternatively, the head-injured trauma patient with a GCS of 12 at presentation that is a 10 now probably needs definitive airway management.
- The table below summarizes the three steps and nine S's. This approach is illustrated in 3 cases below.

SIGNS & SYMPTOMS	
Step 1: Is there evidence of airway obstruction now ?	<ul style="list-style-type: none"> • Complete Obstruction <ul style="list-style-type: none"> ○ Silence without chest rise or ○ See-Saw Chest movement • Partial Obstruction <ul style="list-style-type: none"> ○ Stridor — airway swelling/ compression by hematoma ○ Secretions — saliva, blood ○ Snoring — tongue relaxation ○ Smash — risk of teeth/blood in the airway
Step 2: Is there a risk of anticipated airway obstruction?	<ul style="list-style-type: none"> • Singe or Sputum (carbonaceous) — risk of delayed airway swelling from inhalational burn • Stab or Swelling neck — risk of delayed airway compression from expanding hematoma or neck mass
Step 3: Is there a risk of Aspiration from failure to PROTECT their airway?	<ul style="list-style-type: none"> • Sleepy (low GCS)

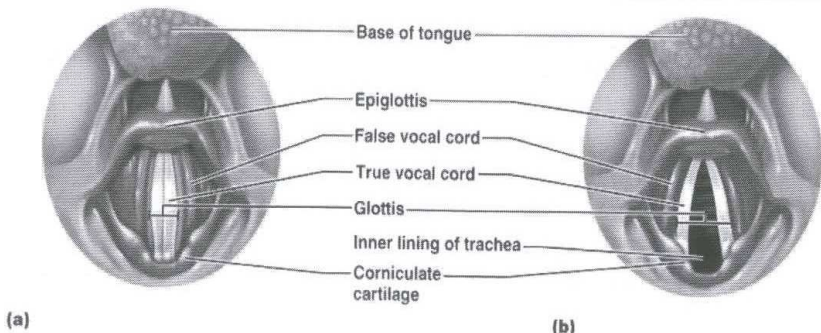


Figure 6.10.1. Vocal cords anatomy

II. CRICOTHYROIDOTOMY

INDICATIONS

- Cricothyroidotomy is indicated upon failure to obtain an airway with traditional methods in the following situations: **CAN'T INTUBATE, CAN'T VENTILATE (CICV) SCENARIO**
 - Trauma causing oral, pharyngeal, or nasal hemorrhage
 - Facial muscle spasms or laryngospasm
 - Uncontrollable emesis
 - Upper airway stenosis or congenital deformities
 - Clenched teeth
 - Tumor, cancer, or another disease process or trauma causing mass effect
- **Airway obstruction indications include the following:**
 - Oropharyngeal edema (eg, anaphylaxis)
 - Foreign body obstruction
- **The following are relative indications for cricothyroidotomy:**
 - Cervical spine immobilization secondary to injury
 - Maxillofacial injuries

CONTRAINDICATIONS

- Ability to secure an airway with less invasive means (Can intubate and/or ventilate)
- Inability to identify landmarks (cricothyroid membrane)
- Underlying anatomical abnormality such as a tumor or severe goiter.
- Tracheal transection.
- Acute laryngeal disease due to infection or trauma.
- Small children under 10 years old (a 12–14-gauge catheter over the needle may be safer)

COMPLICATIONS

EARLY COMPLICATIONS	LATE COMPLICATIONS
<ul style="list-style-type: none"> • Bleeding • Incorrect placement, resulting in possible creation of a false passage through tissue • Subcutaneous emphysema • Obstruction • Esophageal or mediastinal perforation • Aspiration • Vocal cord injury • Pneumothorax • Laryngeal injury • Posterior tracheal wall perforation • Thyroid perforation • Hypercarbia (needle cricothyroidotomy) 	<ul style="list-style-type: none"> • Dysphonia • Infections • Hematoma • Persistent stoma • Scarring • Glottic or subglottic stenosis • Laryngeal stenosis • Tracheoesophageal fistula • Tracheomalacia

PERIPROCEDURAL CARE

- **Equipment**
 - For surgical cricothyroidotomy, materials needed include the following:
 - Antiseptic preparation solution
 - Lidocaine/ Sterile drape/gown/gloves
 - No. 11 scalpel blade/ Syringe (10 mL)
 - Bag-valve mask/ Trousseau dilator/ Tracheal hook

- Tracheostomy tube or endotracheal tube
- **For needle cricothyrotomy**, materials needed include the following:
 - Antiseptic solution/ Lidocaine/ Sterile materials
 - Angiographic catheter (14 gauge or larger)
 - Syringe (10 mL) filled with 5 mL of normal saline
- **For percutaneous cricothyrotomy** using the Seldinger technique, materials needed include a finder needle with a dilator and a guide wire (available in cricothyrotomy kits).
- **PATIENT PREPARATION**
 - In an emergency situation, **there is little time to provide anesthesia**.
 - Often, anesthesia is an unnecessary step that interferes with the acquisition of an emergency airway.
 - If anesthesia is indicated, local anesthesia using 1% lidocaine without epinephrine is appropriate.
 - **The patient should be placed supine** with the neck in the neutral position and medical personnel standing on the patient's right side.
 - **C-spine immobilization** should be applied if indicated.
- **APPROACH CONSIDERATIONS**
 - There are 3 main approaches to cricothyroidotomy: needle cricothyroidotomy, percutaneous cricothyroidotomy using the Seldinger technique, and surgical cricothyroidotomy.

1. NEEDLE CRICOTHYROIDOTOMY

- Needle cricothyroidotomy may be divided into the following steps:
 - Position the patient, apply lidocaine (if indicated), and prepare a sterile field, including cleansing with antiseptic solution.
 - **Identify anatomic landmarks (Cricothyroid Membrane):** Palpate the **thyroid cartilage** (the first prominent landmark on the anterior neck), the **cricoid cartilage** (caudal to the thyroid cartilage), and the **area between them**, which is the cricothyroid space that contains the membrane.
 - With the nondominant hand, stabilize the area using the first and third digits to either side of the thyroid cartilage, leaving the index finger to palpate the membrane.

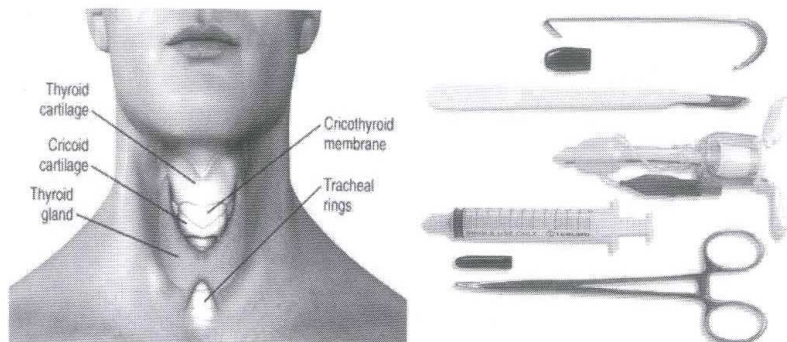


Figure 6.10.2. Landmark and Equipments for Cricothyroidotomy

2. PERCUTANEOUS CRICOTHYROIDOTOMY (SELDINGER)

- Percutaneous cricothyroidotomy using the Seldinger technique may be divided into the following steps:
 - Follow steps 1-4 from needle cricothyroidotomy (see above), substituting a finder needle attached to a syringe for the angiographic catheter.
 - Remove the syringe from the needle, and advance the guide wire through the needle. Remove the needle once the guide wire is in place.
 - Use the scalpel to make a small stab incision in the skin close to the guide wire.
 - Place the dilator into the airway catheter, and insert the 2 devices together over the wire.
 - Remove both the dilator and the guide wire once the airway tube is secured in the trachea.
 - Secure the tube in place with appropriate tape.

3. SURGICAL CRICOTHYROIDOTOMY

- Surgical cricothyroidotomy may be divided into the following steps:
 - Follow steps 1-2 from needle cricothyroidotomy (see above).
 - With the dominant hand, make a midline vertical incision, approximately 3 cm long and skin deep, over the cricothyroid membrane. A midline vertical incision may result in a small amount of venous bleeding but avoids the laterally located vasculature of the neck.
 - Palpate the cricothyroid membrane through the incision, using the index of the nondominant hand. Make a horizontal stab incision through the membrane. A distinct pop will be felt as the scalpel pierces the membrane and enters the trachea.
 - An assistant should insert the tracheal hook at the superior end of the incision and retract the skin and membrane cephalad. Keep the scalpel in place until the tracheal hook is inserted. If the incision is lost, the location can be identified by means of air bubbles produced during exhalation. If the patient is apneic, apply pressure to the anterior chest wall to simulate exhalation and thereby produce air bubbles.
 - Dilate the incision vertically, using the Trousseau dilator with the nondominant hand.
 - With the dominant hand, insert the tracheostomy tube between the 2 blades of the dilator, directing it initially to one side of the patient. Once the tube is through the membrane, rotate it 90° and insert caudally.
 - Remove the obturator, and insert the inner cannula. Lock it into place.
 - Inflate the balloon with 5-10 mL of air. Attach the tube to a BVM and ventilate.
 - Confirm placement through observation of chest rise, auscultation, and assessment of end-tidal CO₂.
 - Remove the tracheal hook, and secure the tube in place.

CHAPTER 11. BASIC AND ADVANCED LIFE SUPPORT



Resuscitation Council (UK)

GUIDELINES
2015

1. BASIC LIFE SUPPORT

- **High quality CRP with minimal interruptions:**
 - Change sequence from "ABC" to "CAB"
 - Chest compressions first then ventilations after first cycle
 - "Look, listen, feel" removed from the algorithm
 - Pulse checks de-emphasised
 - No difference between witnessed and unwitnessed arrest protocols
 - Non-trained bystanders should provide "Hands-only" CPR
 - Compress the chest to a depth of 5–6 cm (adults).
 - Compress the chest at a rate of 100–120/min
 - Complete chest recoil after each compression
 - Minimise interruptions in compressions
 - Avoid excessive ventilation
 - Emphasis on team approach with multiple simultaneous interventions
- **Electrical therapy**
 - Lay people encouraged to use an AED, even without training
 - Initial **biphasic shock** for AF is **150J**
 - Adults with **stable monomorphic VT** - trial synchronised 100J

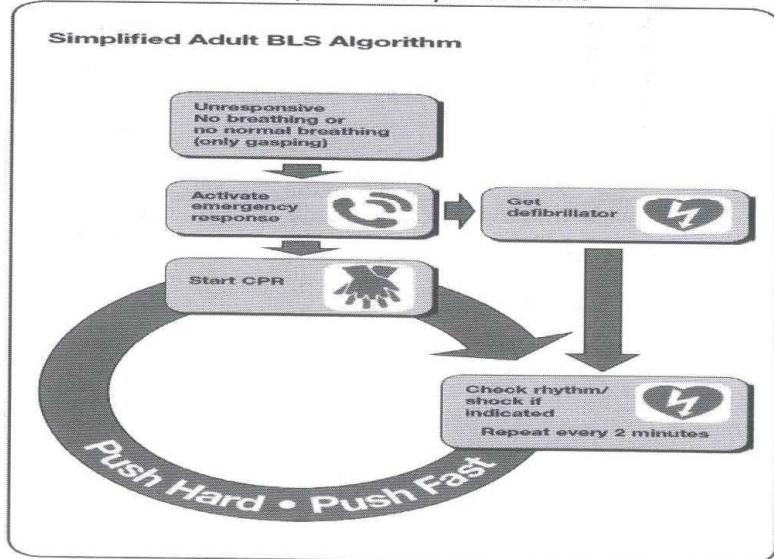


Figure 6.11.1. Adult BLS Algorithm

CHAPTER 12. DEFIBRILLATION & CARDIOVERSION

- **Defibrillation** is nonsynchronized random administration of shock during a cardiac cycle.
- **Cardioversion** is a synchronized administration of shock during the R waves or QRS complex of a cardiac cycle.
- *During defibrillation and cardioversion, electrical current travels from the negative to the positive electrode by traversing myocardium. It causes all of the heart cells to contract simultaneously. This interrupts and terminates abnormal electrical rhythm.*
- *This, in turn, allows the sinus node to resume normal pacemaker activity.*
- **INDICATIONS**
 - **Indications for defibrillation include the following:**
 - Pulseless ventricular tachycardia (VT)
 - Ventricular fibrillation (VF)
 - Cardiac arrest due to or resulting in VF
 - **Indications for electrical cardioversion include the following:**
 - Supraventricular tachycardia (atrioventricular nodal reentrant tachycardia [AVNRT] and atrioventricular reentrant tachycardia [AVRT])
 - Atrial fibrillation
 - Atrial flutter (types I and II)
 - Ventricular tachycardia with pulse
 - Any patient with reentrant tachycardia with narrow or wide QRS complex (ventricular rate >150 bpm) who is unstable (eg, ischemic chest pain, acute pulmonary edema, hypotension, acute altered mental status, signs of shock)
- **CONTRAINDICATIONS**
 - **Dysrhythmias** due to enhanced automaticity, such as in digitalis toxicity and catecholamine-induced arrhythmia.
 - *A homogeneous depolarization state already exists. Therefore, cardioversion is not only ineffective but is also associated with a higher incidence of postshock ventricular tachycardia/ventricular fibrillation (VT/VF).*
 - **Multifocal atrial tachycardia.**
- **ANESTHESIA**
 - Defibrillation is an emergent maneuver and, when necessary, should be promptly performed in conjunction with or prior to administration of induction or sedative agents.
 - Cardioversion is almost always performed under induction or sedation (short-acting agent such as midazolam). The only exceptions are if the patient is hemodynamically unstable or if cardiovascular collapse is imminent.
- **EQUIPMENT**
 - Defibrillators (automated external defibrillators [AEDs], semiautomated AEDs, standard defibrillators with monitors)/ Paddle or adhesive patch
 - Conductive gel or paste, ECG monitor with recorder/ Oxygen equipment, Intubation kit
 - Emergency pacing equipment/ Blood pressure cuff (automatic or manual)
 - Pulse recorder, Oxygen saturation monitor/ Intravenous access, Suction device
 - Code Cart with ACLS (Advanced Cardiovascular Life Support) medications
- **POSITIONING**
 - Paddle placement on the chest wall has 2 conventional positions: **anterolateral and anteroposterior.**

- **IN THE ANTEROLATERAL POSITION:**
 - A single paddle is placed on the **left fourth or fifth intercostal space on the midaxillary line.**
 - The second paddle is placed just to the **right of the sternal edge** on the second or third intercostal space.
- **IN THE ANTEROPOSTERIOR POSITION:**
 - A single paddle is placed to the right of the sternum, as above, and the other paddle is placed **between the tip of the left scapula and the spine.**
- *An anteroposterior electrode position is more effective than the anterolateral position for external cardioversion of persistent atrial fibrillation.*
- *The anteroposterior approach is also preferred in patients with implantable devices, to avoid shunting current to the implantable device and damaging its system.*
- **MONOPHASIC VERSUS BIPHASIC WAVEFORMS**
 - Defibrillators can deliver energy in various waveforms that are broadly characterized as monophasic or biphasic.
 - **Monophasic defibrillation** delivers a charge in **only one direction.**
 - **Biphasic defibrillation** delivers a charge in one direction for half of the shock and in the electrically opposite direction for the second half.
 - Newer defibrillators deliver energy in biphasic waveforms. Biphasic waveform defibrillators deliver a more consistent magnitude of current. They tend to successfully terminate arrhythmias at lower energies than monophasic waveform defibrillators.
- **ENERGY SELECTION FOR DEFIBRILLATION OR CARDIOVERSION**
 - In 2010, the American Heart Association issued guidelines for initial energy requirements for monophasic and biphasic waveforms.
 - **Atrial fibrillation:**
 - 200 Joules for monophasic devices
 - 120-200 Joules for biphasic devices
 - **Atrial flutter:**
 - 100 Joules for monophasic devices
 - 50-100 Joules for biphasic devices
 - **Ventricular tachycardia with pulse:**
 - 200 Joules for monophasic devices
 - 100 Joules for biphasic devices
 - **Ventricular fibrillation or pulseless ventricular tachycardia:**
 - 360 Joules for monomorphic devices
 - 120-200 Joules for biphasic devices

	BROAD COMPLEX TACHYCARDIA	NARROW COMPLEX TACHYCARDIA
IRREGULAR	<ul style="list-style-type: none"> ○ AF with a BBB ○ Pre-excited AF ○ Torsades de pointes 	<ul style="list-style-type: none"> ○ Atrial Fibrillation ○ Atrial flutter with variable block ○ Multifocal Atrial Tachycardia
DC SHOCK	Defibrillation dose, Not synchronised	120-200j biphasic or 200j monophasic
REGULAR	<ul style="list-style-type: none"> ○ VT ○ SVT with BBB ○ Sinus tachycardia with BBB ○ Atrial flutter with BBB 	<ul style="list-style-type: none"> ○ Sinus tachycardia ○ Atrial Flutter ○ Re-entrant SVT
DC SHOCK	100j	50-100j

COMPLICATIONS

- The most common complications are **harmless arrhythmias**, such as **atrial, ventricular, and junctional premature beats**.
- **Serious complications include:**
 - Ventricular fibrillation (VF) resulting from high amounts of electrical energy,
 - Digitalis toxicity,
 - Severe heart disease,
 - Improper synchronization of the shock with the R wave.
 - Thromboembolization is associated with cardioversion in 1-3% of patients,
 - Myocardial necrosis can result from high-energy shocks.
 - Pulmonary edema is a rare complication of cardioversion.
 - Painful skin burns can occur after cardioversion or defibrillation; they are moderate to severe in 20-25% of patients.
 - Allergic reaction to sedation medication is a potential complication.

PRE-PROCEDURE FOR DC CARIOVERSION

- **Gain written consent from the patient:** Risks of sedation; pain (musculoskeletal or minor skin injury); failure (approx. 20%); stroke
- Ensure the area where de-fibrillator pads are to be placed is shaved and dry and that jewellery is removed
- Ensure **cardiac monitoring** is in place
- **Sedation** as necessary

POST-PROCEDURE

- Continue cardiac monitoring
- Post-procedure observations
- Request cardiology review for further investigation, medication and follow up

SPECIAL POPULATIONS

- **CARDIOVERSION IN PATIENTS WITH DIGITALIS TOXICITY**
 - Digoxin overdose or toxicity can present with any type of tachyarrhythmias or bradyarrhythmias.
 - Cardioversion in the setting of digoxin toxicity **is a relative contraindication**.
 - Digitalis sensitizes the heart to the electrical stimulus.
 - Prior to cardioversion, electrolytes should be normalized. Cardioversion may cause **additional arrhythmias**, especially **ventricular fibrillation**.
- **CARDIOVERSION IN PATIENTS WITH PERMANENT PACEMAKERS/ICDS**
 - Cardioversion in patients with permanent pacemaker/ICD should be performed with extra care. Improper technique may **damage the device, lead system, or myocardial tissue**, resulting in device malfunction.
 - The electrode paddle or patch should be at least **12 cm from the pulse generator and anteroposterior** paddle position.
 - The lowest amount of energy should be used during cardioversion, based on the patient's clinical condition. After cardioversion, the pacemaker/ICD should be interrogated to ensure normal function of the device.
- **CARDIOVERSION DURING PREGNANCY**
 - Cardioversion can be performed safely in pregnant women.
 - **The fetal heart rate** should be monitored during the procedure using fetal monitoring techniques.

CHAPTER 13. TEMPORARY PACEMAKERS

I. NON-INVASIVE TRANSCUTANEOUS PACING

- There are two types of artificial pacemaker: **Temporary and Permanent**.
 - **Permanent (epicardial) pacemakers** are implanted by means of a surgical procedure and are used to treat permanent conduction problems.
 - **Temporary pacemakers** are used in emergency situations for transient conduction disturbances or prophylactically for anticipated dysrhythmias.
 - Temporary pacemakers may be **invasive** (transvenous) or **non-invasive** (transthoracic).
 - **Temporary non-invasive pacemakers** are typically available to clinicians as part of a cardiac resuscitation system, complete with defibrillation, cardioversion and monitoring capabilities.
- **INDICATIONS FOR NON-INVASIVE PACING**
 - **Hemodynamically unstable bradycardia** not responding to Atropine
 - **Acute MI associated with:**
 - Symptomatic sinus bradycardia
 - Mobitz type II second-degree AV block
 - Third-degree AV block
 - New LBBB with 1st degree AV Block
 - Alternating LBBB and RBBB
 - New Bifascicular block (RBBB+LAD)
 - **Termination of SVT or VT** by overdrive pacing
 - **Used on standby in situations** when the patient is clinically stable yet may quickly decompensate or become unstable
 - **Used when invasive pacing is undesirable**
 - **Drug overdose or patients PEA** due to acidosis or electrolyte abnormalities
- **INDICATIONS FOR USE OF NTP IN CHILDREN**
 - *Bradycardias from surgically acquired AV blocks*
 - *Congenital AV blocks*
 - *Viral myocarditis*
 - *Heart block secondary to toxin or drug overdose*
 - *Permanent pacemaker generator failure.*
- **CONTRAINDICATIONS TO NTP**
 - **Severe hypothermia** since the heart is unable to respond to the electrical stimulus
 - **Confusion:** since there may be difficulty keeping electrodes securely in place and the discomfort will increase the agitation.
- *Non-invasive pacing is used on a temporary basis until the patient is stabilized and either an adequate intrinsic rhythm has returned or a transvenous pacemaker is inserted, whether temporary or permanent.*
- Transcutaneous pacing should be initiated without delay when there is impairment in the conduction system resulting in a high-degree block (e.g., Mobitz type II second-degree block or third-degree AV block). While waiting for the pacemaker device, **atropine should be considered**. In an emergency, if there is no intravenous access, the atropine is not effective or the patient is severely symptomatic, **NTP should be begun immediately by the trained nurse or physician.**

- NTP can be set up ready to use in patients who are clinically stable yet may quickly decompensate.
- **SYNCHRONOUS/ASYNCHRONOUS MODES**
 - Most defibrillators have both fixed rate and synchronous pacing.
 - **Synchronous pacing** is a **demand mode**, in which the pacer fires only when no complex is sensed for a predetermined amount of time.
 - Pacing generally should be started in the synchronous mode to coordinate the efforts of the cardiac resuscitation system's pacemaker with the patient's own cardiac electrical activity.
 - **In the fixed rate (asynchronous) mode**, the non-invasive pacemaker delivers an electrical stimulus at preset intervals, independent of intrinsic cardiac activity.
- **METHOD OF INSERTION AND/OR USE**
 - Obtain consent
 - Consider sedation and analgesia
 - Prepare skin
 - Connect ECG leads
 - Place pads in AP position (black on anterior chest, red on posterior chest)
 - Set pacemaker to demand
 - Rate: set rate to **> 30bpm above patient's intrinsic rhythm**
 - **Set initial current to 70 mA**
 - Start pacing and increase current until pacing rate captured on monitor: each pacemaker is followed by wide QRS and T.
 - **Recheck the pulse**
 - If pacing rate not captured at a current of **120-130mA** -> resite electrodes and repeat the above.
 - Once pacing captured, set current at **5-10mA above threshold**
- **ELECTRODES**
 - Non-invasive pacing can cause discomfort for patients and can be quite painful.
 - Pain is a function of the current delivered per unit of skin surface area.
 - Electrodes with a large surface area minimize pain sensation. Most commercially available electrodes are 80-100 cm².
- **ANTERIOR - POSTERIOR PLACEMENT**
 - It is preferred for external, non-invasive pacing.
 - It may provide improved capture and will not interfere with defibrillation if required.
 - **Place negative electrode on left anterior chest halfway between the xiphoid process and left nipple**, with the upper edge of the electrode below the nipple line.
 - **Place positive electrode on left posterior chest beneath the scapula and lateral to the spine.**
- **ANTERIOR - LATERAL PLACEMENT**
 - Requires little patient movement and allows for easy monitoring or defibrillation during transport.
 - **Place negative electrode on left chest midaxillary** around the fourth interspace.
 - **Place positive electrode on right chest**, subclavicular area.

CHAPTER 14. LARGE JOINT EXAMINATION

I. KNEE CLINICAL EXAMINATION

HISTORY

• PAIN CHARACTERISTICS

- The patient's description of knee pain is helpful in focusing the differential diagnosis.
- It is important to clarify the **characteristics of the pain**:
 - Its onset (rapid or insidious), location (anterior, medial, lateral, or posterior knee), duration, severity, and quality (e.g., dull, sharp, achy).
 - Aggravating and alleviating factors also need to be identified.
 - If knee pain is caused by an acute injury, the physician needs to know whether the patient was able to continue activity or bear weight after the injury or was forced to cease activities immediately.

• MECHANICAL SYMPTOMS

- The patient should be asked about mechanical symptoms, such as locking, popping, or giving way of the knee.
- A history of **locking** episodes suggests a **meniscal tear**.
- A sensation of **popping** at the time of injury suggests **ligamentous injury**, probably complete rupture of a ligament (third-degree tear).
- Episodes of **giving way** are consistent with some degree of **knee instability** and may indicate patellar subluxation or ligamentous rupture.

• EFFUSION

- The timing and amount of joint effusion are important clues to the diagnosis.
- **Rapid onset** (within two hours) of a large, tense effusion suggests rupture of the anterior cruciate ligament or fracture of the tibial plateau with resultant hemarthrosis.
- Whereas **slower onset** (24 to 36 hours) of a mild to moderate effusion is consistent with meniscal injury or ligamentous sprain.
- **Recurrent knee effusion** after activity is consistent with meniscal injury.

• MECHANISM OF INJURY

- The patient should be questioned about specific details of the injury:
 - If the patient sustained a direct blow to the knee?
 - If the foot was planted at the time of injury?
 - If the patient was decelerating or stopping suddenly?
 - If the patient was landing from a jump?
 - If there was a twisting component to the injury, and if hyperextension occurred?
- **Anterior force** applied to the proximal tibia with the knee in flexion (e.g., when the knee hits the dashboard in an automobile accident) can cause **injury to the PCL**.
- **The medial collateral ligament** is most commonly injured as a result of **direct lateral force to the knee** (e.g., clipping in football).
- Conversely, a **medial blow** that creates a varus load can injure the **lateral collateral ligament**.
- Quick stops and sharp cuts or turns create significant deceleration forces that can sprain or **rupture the ACL**.
- **Hyperextension** can result in **injury to the ACL or PCL**.
- **Sudden twisting or pivoting motions** create shear forces that can injure the **meniscus**.

- A combination of forces can occur simultaneously, causing injury to multiple structures.

• **MEDICAL HISTORY**

- The patient should be asked about previous attempts to treat knee pain, including the use of medications, supporting devices, and physical therapy.
- The physician also should ask if the patient has a history of gout, pseudogout, rheumatoid arthritis, or other degenerative joint disease.

PHYSICAL EXAMINATION

• **INSPECTION AND PALPATION**

- **Look:** The physician begins by comparing the painful knee with the asymptomatic knee and inspecting the injured knee for erythema, swelling, bruising, and discoloration.
- **Feel:** for pain, warmth, and effusion.
- **Move:** Range of motion should be assessed by extending and flexing the knee as far as possible (**Normal range of motion: Extension= zero degrees; Flexion= 135 degrees**).

1. PATELLOFEMORAL ASSESSMENT

- An evaluation for effusion should be conducted with the patient supine and the injured knee in extension. **The suprapatellar pouch** should be milked to determine whether an **effusion** is present. Patellofemoral tracking is assessed by observing the patella for smooth motion while the patient contracts the quadriceps muscle.
- The presence of crepitus should be noted during palpation of the patella.

A. PATELLAR APPREHENSION TEST

- With fingers placed at the medial aspect of the patella, the physician attempts to sublux the patella laterally. If this maneuver reproduces the patient's pain or a giving-way sensation, **patellar subluxation** is the likely cause of the patient's symptoms.

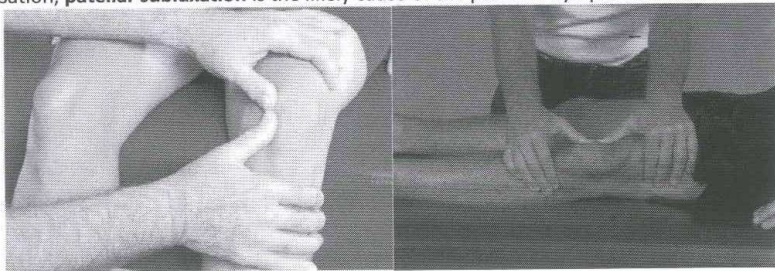


Figure 6.14.1. Patella Apprehension Test

- Both the superior and inferior patellar facets should be palpated, with the patella subluxed first medially and then laterally.

2. ANTERIOR CRUCIATE LIGAMENT

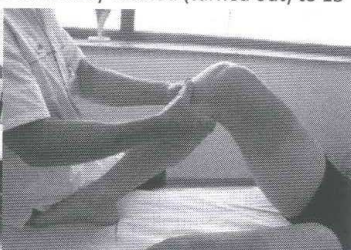
A. ANTERIOR DRAWER

- **Description**
 - The Anterior Drawer test examines for **any tearing or laxity of the ACL ligament**.
- **Maneuver**
 - Have the patient lying on their back with their knee bent as close to 90° as possible, with the foot resting on the table. Place both hands behind tibia and pull the tibia forward, using a force between 15-20 lbs.

- The test can also be performed with the foot externally rotated (turned out) to 15°.

Anterior Drawer Test

Anterior Cruciate Ligament Insufficiency



○ Positive Findings

- **Increased anterior movement of the tibia** on the injured side compared to the non-injured side is considered to be a positive test.
- Up to 3 mm of forward movement of the tibia is considered normal.
- The Grading: **Grade 1 = 5 mm, Grade 2 = 5 to 10 mm, Grade 3 > 10 mm.**

B. THE LACHMAN TEST

- It is another means of assessing the integrity of the anterior cruciate ligament.
- The test is performed with the patient in a supine position and the injured knee flexed to 30 degrees. The physician stabilizes the distal femur with one hand, grasps the proximal tibia in the other hand, and then attempts to sublux the tibia anteriorly.
- **Lack of a clear end point** indicates a positive Lachman test.

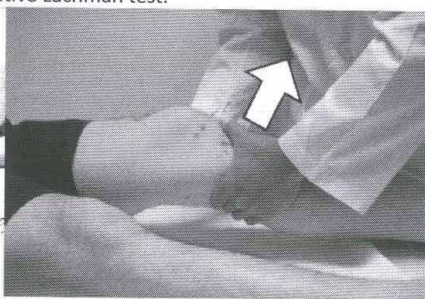
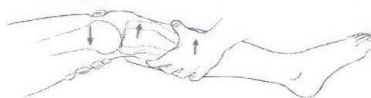
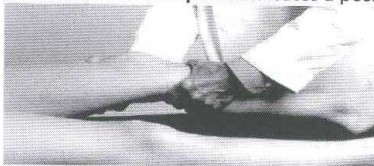


Figure 6.14.2. Lachman Test

3. POSTERIOR CRUCIATE LIGAMENT

A. POSTERIOR DRAWER

○ Description:

- The posterior drawer test is used to **examine the Posterior Cruciate Ligament (PCL).**

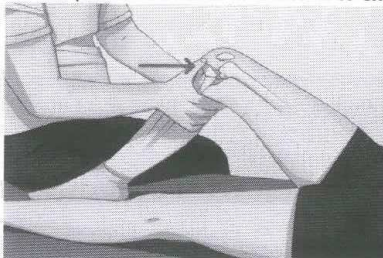


Figure 6.14.3. Posterior drawer test

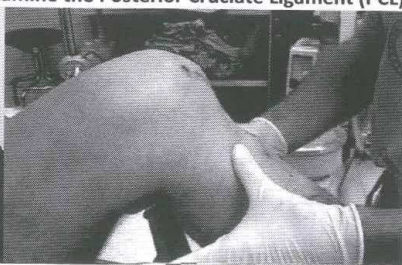


Figure 6.14.4. PCL Injury

- **Maneuver**

- Have the patient lying on their back with their knee bent as close to 90° as possible with their foot resting on the table.
- Place both hands behind the tibia, and push backwards on the proximal shin/tibia looking for instability backwards. Use a force between 15-20 lbs.

- **Positive Findings**

- Upon application of a posterior force to the upper shin, an increase in backwards motion in comparison to the other side is indicative of a positive test.

4. MEDIAL & LATERAL COLLATERAL LIGAMENTS

A. VALGUS STRESS TEST

- **Description**

- The valgus stress test **checks for medial joint laxity**, which usually represents an injury to the **medial collateral ligament (MCL)**.

- **Maneuver**

- Have patient lie on their back. Position one hand at the joint line on the outer part of the knee. Have the other hand fixed on the ankle of the affected side.
- Flex the knee between 20° and 30° and apply a **medial or valgus** force to the knee.
- In order to test the MCL, as well as the posterior medial capsule, the test can be repeated at 0° with the knee in full extension.

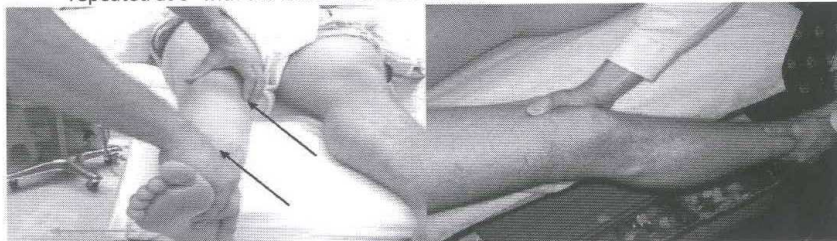


Figure 6.14.5. VALGUS TEST (Externally; checks MCL) Figure 6.14.6. VARUS TEST (Internally; checks LCL)

- **Positive Findings**

- A positive test demonstrates **increased medial joint laxity** compared to the unaffected side. Grading system: **Grade 1= 5mm, Grade 2= 5 to 10mm, Grade 3= >10 mm.**

B. VARUS STRESS TEST

- **Description**

- The varus stress test checks for joint laxity on the outside of the knee, which usually represents an injury to the **lateral collateral ligament (LCL)**.

- **Maneuver**

- With the patient lying on their back, position one hand at the joint line on the outer part of the knee. Fix the other hand on the ankle of the affected side.
- Flex the knee between 20° and 30° and apply a **lateral or varus** force to the knee.
- This can be done either by reaching over the top of the knee, or by approaching the patient from the inside aspect of the knee with the leg off to the side.
- The test can also be repeated at 0° with the knee in full extension.

- **Positive Findings**

- A positive test demonstrates **increased lateral joint laxity** compared to the unaffected side.
- Grading system: **Grade 1= 5mm, Grade 2= 5 to 10mm, Grade 3= >10 mm.**

5. MENISCI

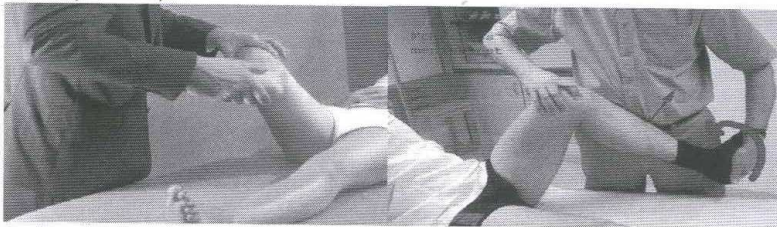
A. MCMURRAY'S TEST

○ Description

- This test checks for **meniscal tears** and other internal derangement in the knee.

○ Maneuver

- With the patient supine, and their hip and knee bent to 90°, grasp the heel in one hand. Place the other hand over the knee, with the thumb and fingers on the joint line.
- Gently rotate the tibia with the heel internally rotated with a mild valgus force (for the lateral compartment) and externally rotated with a mild varus force (for the medial compartment).



○ Positive Findings

- **Painful clicking along the joint line**, or **any pain over the joint line** that reproduces the patient's symptoms is considered to be a positive test.

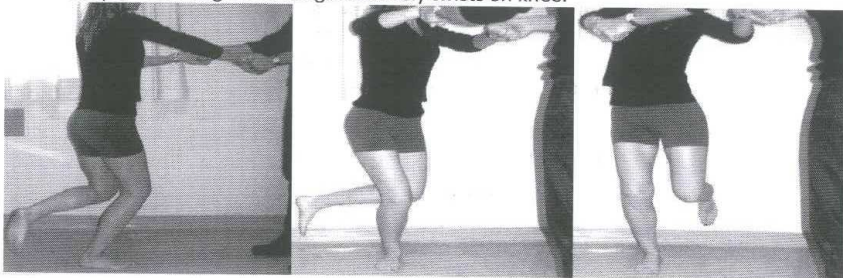
B. THESSALY'S TEST

○ Description

- This functionally tests **meniscus tears in the standing position**. Since bending and twisting movements while weight bearing often reproduce pain from meniscus tears, this test recreates the exacerbating movements.

○ Maneuver

- Have the patient stand on one foot with the foot flat on the floor.
- Hold the patients hand for support and have them initially bend on the standing knee to 5° of flexion. Ask the patient to twist at the knee, making sure they are internally and externally rotating at the knee rather than at the pelvis or back.
- Check for any reproduction of pain symptoms. Next, have the patient bend the knee deeper to 20° degrees and again actively twists on knee.



○ Positive Findings

- The twisting movement will reproduce pain of a meniscal injury.
- The pain is typically localized to joint line, and patients typically have more pain with the knee bent at 20° rather than 5°.

II. SHOULDER PHYSICAL EXAMINATION

• EXAMINATION

- Inquire about the patient's hand dominance, as well as their occupation and recreational activities. Establish their chief complaint: pain, instability, weakness, or loss of range of motion. Establish an approximate timeline for when the injury occurred and what event or mechanism, if any, lead to the injury or onset of symptoms. For patients who report a dislocation, it should be asked what position the arm was in at the time of the dislocation, and what the frequency of dislocations or subluxations were. Establish what type of activities of daily living the patient can and cannot perform. Such activities include simple everyday tasks like getting dressed, lifting an object overhead, sleeping on the shoulder, brushing your teeth, combing your hair, putting on shoes, and carrying or lifting objects like groceries.

• PALPATION

- Bony structures to palpate should include: the sternoclavicular joint, the clavicle, the acromioclavicular joint, the coracoid process, the borders of the scapula, and the greater and lesser tuberosities of the humerus.
- Soft tissue landmarks should include: the subacromial bursa, the supraclavicular fossa, the long head of the biceps tendon, the trapezius, and other associated muscles and tendons.

• RANGE OF MOTION

- **Active range of motion** performed by the patient is typically assessed first, and can be affected by both pain and motor function.
- The patient can be either seated or standing during the assessment, and movements to be tested should include forward flexion, extension, internal/external rotation, and abduction/adduction.
- **Passive range of motion** is performed by the clinician with the patient seated or supine in the same planes previously stated. This is used to isolate motion for an accurate evaluation of soft tissue.
 - *Normal flexion: 0° to 170-180°*
 - *Normal extension is said to be 60°.*
 - *Normal internal rotation is said to be 90°*
 - *Normal external rotation is around 60-70°.*
 - *Normal adduction is typically 30°*
 - *Normal Abduction motion can range from 0° to 180°*
- An example of limited passive range of motion can be seen in cases of frozen shoulder.
 - **Supraspinatus:** abduction >>> Empty can test
 - **Subscapularis:** internal rotation >>> Lift-off test
 - **Infraspinatus and Teres minor:** external rotation >>> External rotation test

1. FROZEN SHOULDER: EXTERNAL ROTATION

- To improve range of motion, special exercises such as Codman's Pendulum can be performed to help relax the muscles around the shoulder, reduce pain, and increase motion.

A. CODMAN'S PENDULUM

- Have the patient standing in a relaxed position, and tell them to swing their weak arm in a circular motion while keeping their shoulder nice and relaxed.
- Be sure they swing their arm in both the clockwise and counterclockwise directions.

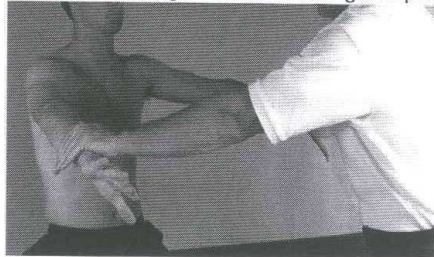


Codman Pendulum Circles

2. ROTATOR CUFF STRENGTH TESTING:

A. EMPTY CAN TEST

- **Description:** The empty can test is used to evaluate the strength and integrity of the **supraspinatus muscle and tendon**.
- **Maneuver:**
 - Have the patient stand with their shoulder abducted to 90° and horizontally adducted forward 30° with the thumbs pointing down towards the floor, as if they are pouring out a can. Ask the patient to maintain this position. Proceed to apply downward resistance to the patient's forearm.
 - A variation of this test can be done at 30° abduction instead of 90°, where the supraspinatus should function in relative isolation.
- **Positive findings:** Decreased strength or pain on resisted testing.



B. EXTERNAL ROTATION

- **Description:** The external rotation test examines the **strength of the infraspinatus and teres minor**.
- **Maneuver:**
 - With the patient's arms at their side, externally rotated 45° and elbow flexed to 90°, the examiner applies an internal rotation moment to assess the strength of the external rotators.
- **Positive Findings:** Decreased strength or pain on resisted testing. Significant weakness of the infraspinatus may be indicative of **suprascapular nerve palsy**, where the infraspinatus become denervated. This can be due to trauma, ganglion cyst or illness.

C. LIFT-OFF TEST

- **Description:** The **lift off test** evaluates the muscular strength of the **subscapularis**.
- **Maneuver:** With the patient seated or standing, have them internally rotate their arm behind their back.
- Then ask the patient to lift the back of their hand off their lower back.

- If they are unable to complete this task, apply resistance to the palm to assess the strength of the subscapularis.
- **Positive findings:** Inability to lift the dorsum of hand off the back.

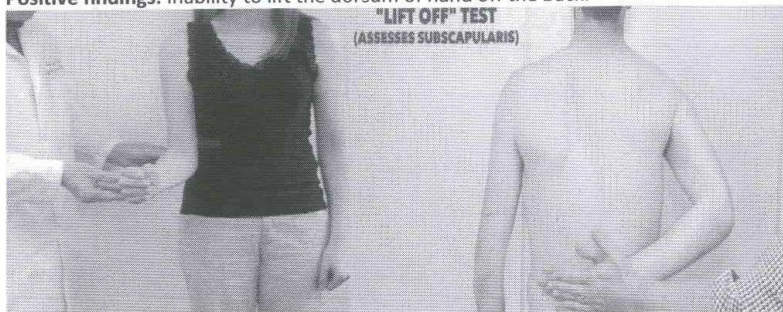


Figure 6.14.7. External rotation test

Figure 6.14.8. Lift-off test

3. IMPINGEMENT/ROTATOR CUFF SPECIAL TESTS:

A. NEER'S IMPINGEMENT

- **Description:** The Neer impingement test assesses the presence of impingement of the rotator cuff, **primarily the supraspinatus**, as it passes under the subacromial arch during forward flexion.
- **Maneuver:** Stabilize the scapula with one hand while applying passive forced flexion of the arm.
- **Positive findings:** Pain in the anterior shoulder or reproduction of the patient's symptoms.



Figure 6.14.9. Neer's Test

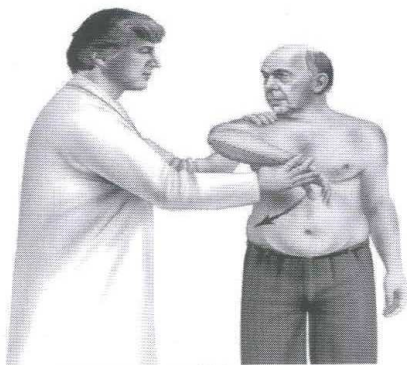


Figure 6.14.10. Hawkin's Kennedy Test

B. HAWKIN'S KENNEDY IMPINGEMENT TEST

- **Description:** The Hawkin's test is used to evaluate **impingement of rotator cuff and subacromial bursa**.
- **Maneuver:** The patient is seated or standing and with their arm forward flexed to 90° and their elbow bent to 90°. Stabilize the top of the shoulder while internally rotating the arm at the forearm.
- **Positive Findings:** Pain in the anterior shoulder or reproduction of the patient's symptoms with the test.

4. INSTABILITY SPECIAL TESTS

A. LOAD AND SHIFT TEST

- **Description:** The Load and Shift test examines integrity of shoulder stability in the anterior and posterior directions.
- **Maneuver:** Have the patient seated or supine with their arm relaxed and resting at their side. Grasp the head of the humerus with thumb and fingers and apply an anterior and posterior glide from the resting position.
- **Positive Findings:** Excessive gliding of the humeral head is considered to be a positive test.



Figure 6.14.11. Load and Shift Test

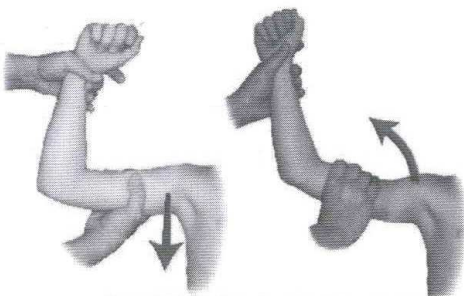


Figure 6.14.12. Apprehension Relocation Test

B. APPREHENSION RELOCATION

- **Description:**
 - The apprehension test, described by Row and Zarin, tests for anterior instability of the shoulder.
 - The relocation test, described by Jobe, is used in conjunction with the apprehension test to distinguish between anterior instability and primary impingement of the shoulder.
- **Maneuver:**
 - To perform the apprehension test, have the patient supine, with their arm abducted and elbow flexed to 90°. Gently externally rotate the arm.
 - Once the patient becomes apprehensive or complains of pain, proceed with the relocation and surprise test by applying a posterior force to the humeral head.
- **Positive Findings:**
 - For the apprehension test, the patient may complain of pain or be apprehensive that their arm may dislocate as it is externally rotated.
 - The relocation test is positive if the symptoms of apprehension reduce, or if the clinician is able to externally rotate the shoulder further without any increase in pain or apprehension. If the symptoms persist following the posterior directed force, the pain is associated with primary impingement and not anterior shoulder instability.

C. SULCUS SIGN

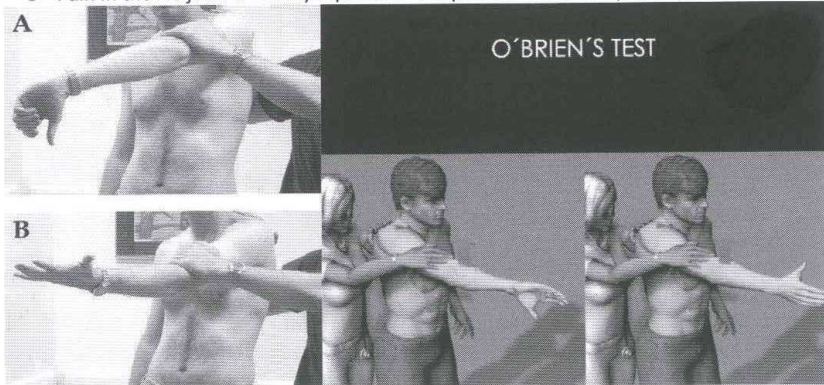
- **Description:** The sulcus sign tests for inferior instability caused by laxity of the inferior glenohumeral ligament complex.
- **Maneuver:** Have the patient seated with their arm resting at their side. Grasp the patient's upper arm and apply a distal force to it.

- **Positive Findings:** Increased inferior movement of the humeral head or the visible development of a sulcus at the glenohumeral joint are positive findings.
- A positive test can often suggest that the patient has multidirectional instability, especially if there are other signs of joint instability.

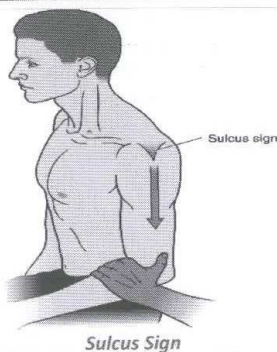
5. LABRAL SPECIAL TESTS

A. O'BRIEN'S TEST

- **Description:** This test examines the integrity of the glenoid labrum and the acromioclavicular joint.
- **Maneuver:** With the patient seated or standing, instruct the patient to raise their arm into 90° of forward flexion with their elbow extended, and then adduct their arm 10-15°. Have the patient internally rotate their arm and point their thumb down to the ground. Apply a downward force to the arm. Then instruct the patient to externally rotate their arm and point their thumb towards the ceiling. Again, apply a downward force.
- **Positive Findings:** Positive findings for labral pathology occur when the first test reproduces pain, while the second test decreases or eliminates pain. The pain associated with labral tears is described as being deep in the shoulder. Pain situated over the acromioclavicular joint is associated with acromioclavicular joint pathology such as osteoarthritis or a shoulder separation, rather than labral pathology.
- Pain in the AC joint is usually equal with the palm down or the palm up.



Shoulder		Tests
Rotator cuff	Supraspinatus	○ Empty can test
	Subscapularis	○ Lift-off test
	Infraspinatus and teres minor	○ External rotation test
	Impingement	○ Neer's test ○ Hawkin Kennedy test
Stability		○ Load & shift test ○ Apprehension & Relocation test ○ Sulcus sign
Labral test		○ O'Brien's test



III. ANKLE PHYSICAL EXAMINATION

• EXAMINATION

- Assessment of gait pattern, standing posture, and shoe wear pattern. Any obvious gross deformity, malalignment, or atrophy should also be observed and noted.

• PALPATION

- Bony structures to palpate: shaft of tibia and fibula, traveling down the borders of both the medial and lateral malleoli.
- Palpation of the neck and dome of the talus should also be performed. This can be done by inverting and everting the foot, and palpating just anterior to the medial and lateral malleoli. Soft tissue palpation should include all the ligamentous structures: the anterior talofibular ligament, the posterior talofibular ligament, the calcaneofibular ligament, the deltoid ligament complex, and the anterior tibiofibular syndesmosis.
- Palpation of the muscle tendons: The peroneus longus and brevis tendon can be palpated as ***it passes posterior to the lateral malleolus and courses below the distal pole towards the base of the fifth metatarsal.***
- On the medial aspect of the ankle, palpation of the posterior tibialis, flexor digitorum longus, and flexor hallucis longus can be done. These three tendons pass posterior to the medial malleolus.
- Finally, along the anterior aspect of the ankle, the body and tendon of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus can be palpated.

• RANGE OF MOTION

- There are **four main motions** that occur at the ankle joint: **dorsiflexion, plantar flexion, inversion, and eversion.**
- Range of motion **should always be compared bilaterally** and any deficits should be noted. Limitation of motion may be a result of pain, swelling, or scar tissue from a chronic injury. Finally, resistive range of motion should be tested to assess for any muscular weaknesses or injuries.

Tests integrity of
calcaneofibular ligament

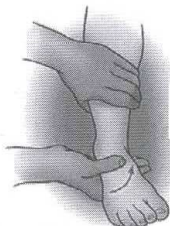
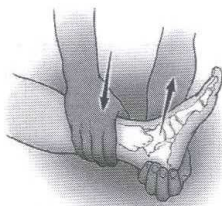
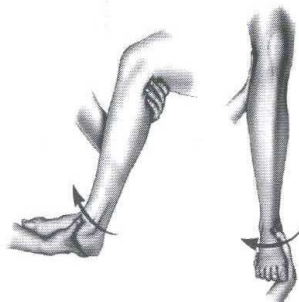


Figure 6.14.13. Talar Tilt shift

Tests integrity of anterior
talofibular ligament



14. Anterior Drawer test



15. Kleiger's Test

1. TALAR TILT TEST

- **Description:** The Talar Tilt Test is a ligamentous stress test that examines **the integrity of the lateral ankle ligaments**, particularly the **calcaneofibular ligament.**
- **Maneuver:** Have the patient in the seated position, with their knee bent and foot in a neutral or slightly dorsiflexed position.
- Stabilize the distal tibia with one hand while applying an **inversion force to the foot.**

- **Positive Findings:** Positive findings include any pain in the ankle or increased joint laxity. Depending on the positioning of the ankle, pain may be experienced over either the calcaneofibular ligament or the anterior talofibular ligament.

2. ANTERIOR DRAWER

- **Description:** The anterior drawer test is used to examine the **integrity of the anterior talofibular ligament**, which is frequently injured during an **inversion ankle sprain**.
- **Maneuver:** Have the patient seated with their knee bent and their ankle in a neutral position at 0° or 90° to the leg. Stabilize the distal tibia with one hand, while grasping the heel with the other hand. Apply an anterior force to the heel. This test should be performed bilaterally to compare for differences in anterior translation.
- **Positive Findings:** Pain or increased joint laxity in the injured ankle **indicates disruption of the anterior talofibular ligament**. A dimple may also be visually seen by the clinician while performing this test.

3. EXTERNAL ROTATION OR KLEIGER' S TEST

- **Description:** The test is used to help **identify syndesmotomic injuries**.
- **Maneuver:** Have the patient seated with their knee bent on the exam table. Stabilize the distal tibia while externally rotating the foot. External rotation of the talus applies pressure to the lateral malleolus, causing a widening of the tibiofibular joint.
- **Positive findings:** Increased external rotation of the foot when compared bilaterally, or any pain in the anterolateral ankle joint is considered to be a positive finding.

4. THOMPSON' S TEST

- **Description:** This test is utilized to evaluate the **integrity of the heel cord**.
- **Maneuver:** Have the patient lying prone on a table with their foot extended off the edge. Squeeze the calf muscle at position slightly distal to the place of widest girth. Examine the movement at the foot.
- **Positive Findings:** A positive test occurs when **the calf is squeezed and no plantar movement occurs at the foot**. This indicates **Achilles tendon rupture**.

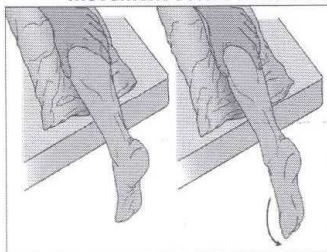


Figure 6.14.16. Thompson's test

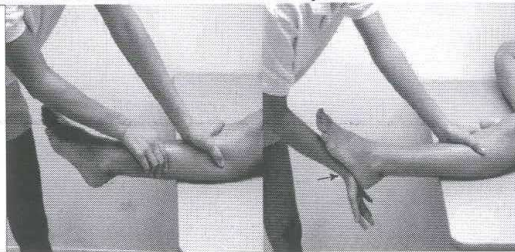


Figure 6.14.17. Compression test

5. COMPRESSION TEST

- **Description:** This test examines **the integrity of the distal tibiofibular joint**. It can also **assess for fractures of the tibia and fibula**.
- **Maneuver:** Have the patient sitting supine with their foot on the table. Grasp the mid-calf and squeeze the tibia and fibula together. Gradually move distally towards the ankle while continuing to apply the same amount of pressure.
- **Positive findings:** Any pain in the lower leg may be indicative of a fracture or syndesmotomic sprain.

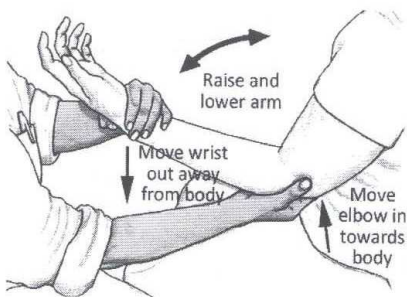
CHAPTER 15. REDUCTION OF DISLOCATION/ FRACTURE

I. SHOULDER RELOCATION TECHNIQUES

- Early reduction is recommended to be performed when **dislocation** has occurred, so to reduce the amount of muscle spasm that must be overcome and minimise the amount of stretch and compression of neurovascular structures.
- Classical techniques still taught include; **Kocher, Hippocratic, Stimson's and Milch**; many of the newer techniques are variations of the classics.
- Choice of technique depends on the experience and preference of the Doctor, facilities available, number of assistants available, time available and the patient's condition.

1. KOCHER'S METHOD

- Flexion of the elbow to a right angle
- Traction in the line of the humerus
- External rotation of the arm: this brings the head of the humerus to face forwards
- The elbow is pulled across the body: this adducts the humerus and disengages the humeral head
- Internal rotation of the arm: this lets the humeral head fall back into the glenoid
- **It is necessary to x-ray the shoulder to confirm the reduction.**
- **Complications include:**
 - *Nerve damage (greater risk than other techniques)*
 - *Fracture of the humerus*
 - *Tearing of the subscapularis muscle*
 - *Damage to the axillary vein*



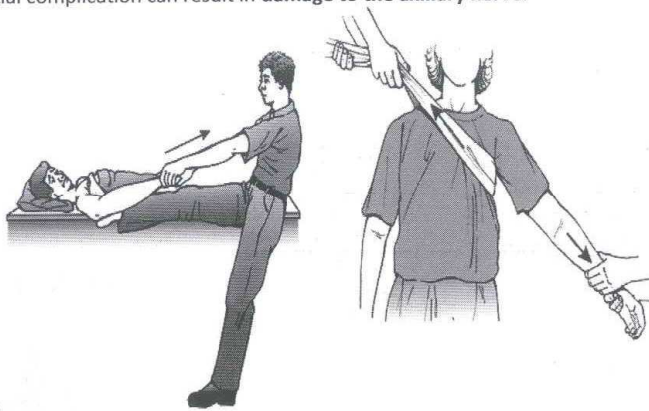
2. MILCH TECHNIQUE

- The surgeon stands on the same side as the affected arm whilst the patient lies in a supine position. The surgeon's fingers are placed over the affected shoulder, to steady the displaced humeral head the thumb is braced against it.
- Next the surgeon's other hand gently abducts and externally rotates the patient's arm into an overhead position, whilst fixing the humeral head so that it does not move from its dislocated position. The surgeon now gently pushes the humeral head back into the glenoid fossa with their thumb.
- The Milch Technique can also be done in the **prone position**:
 - With the patient prone on a table, pillows are placed under the pectoral muscles of the involved shoulder, the arm is allowed to hang freely.
 - Reduction from relaxation can occur spontaneously in this position.

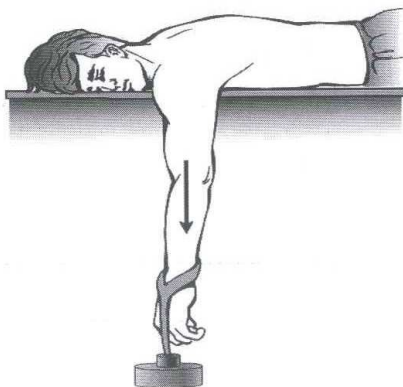


3. HIPPOCRATIC METHOD

- Hippocratic Method begins with the patient supine, the surgeon grasps the affected side at the hand and forearm.
- The stockinged heel of the surgeon is placed in the axilla (not pressed hard) this acts as a fulcrum whilst the arm is adducted.
- Potential complication can result in **damage to the axillary nerve**.

**4. STIMSON'S METHOD**

- Stimson's Method usually requires the patient to have a powerful anagelsic beforehand, and has the patient prone on a table with the affected arm hanging down in forward flexion.
- A sandbag is placed under the clavicle on the affected side, and an approx. 10lb weight is applied to the wrist on the affected side.
- The spasming muscles eventually relax and the joint normally reduces spontaneously

**5. OTHER TECHNIQUES**

- Spaso technique
- Eskimo technique
- Manes method

II. ANKLE RELOCATION

• INDICATIONS

- Traumatic ankle dislocation not associated with neurovascular compromise
- Traumatic ankle dislocation associated with neurovascular compromise
- Subtalar dislocations are rare (<2% of large-joint dislocations) and are the result of high-force mechanisms of injury directed at the forefoot.
 - *Of note, 10-20% of subtalar dislocations are irreducible by closed methods and require operative intervention.*
 - *Radiographs of the ankle, including anteroposterior (AP), lateral, and mortise views, may quickly and reliably differentiate between ankle and subtalar dislocations.*

• CONTRAINDICATIONS

- Failed attempts at closed reduction despite optimal conditions
- Radiographic evidence of a subtalar, rather than ankle, dislocation
- Clinical evidence of a subtalar, rather than ankle, dislocation in the absence of radiographic studies
- *As noted, it is essential to differentiate an ankle injury with dislocation from a subtalar dislocation. An attempt to reduce a subtalar dislocation with ankle reduction techniques is likely to be unsuccessful and may lead to further injury of the involved articular structures.*

PERIPROCEDURAL CARE

• Patient Education and Consent

- Consent for **both the procedure and sedation** should be obtained from the patient or the patient's representative (eg, a family member).
- Explanations of the following should be provided:
 - Reason for performing the procedure (suspected diagnosis)
 - Risks and benefits of the procedure, as well as any alternatives to the procedure
 - Risks and benefits of alternatives to the procedure
 - Risks and benefits of not undergoing the procedure

• Preprocedural Evaluation

- Obtain and document a thorough **preprocedural history**, including the following:
 - History of prior injuries and surgeries, Mechanism of trauma
 - Amount of time elapsed since the traumatic event
 - Description of the presenting symptoms,
 - Prior medical allergies and reactions
 - Any subjective loss of strength or sensation
 - Patient's age in reference to skeletal maturity
- Perform and document a **thorough physical examination**, with particular attention to the following:
 - Ecchymoses, Swelling, Pallor, Abrasions and lacerations, Paresthesias
 - Weakness, Notable deformities of the ankle or foot
 - Presence and character of the dorsalis pedis and posterior tibial pulses
 - Exact position in which the ankle and distal foot are held
 - Comparison examination of the contralateral ankle
- Emphasis should be placed on assessing the **neurovascular status of the distal foot**.
- Carefully explore all areas of skin overlying the ankle joint for dermal compromise that may make the injury an open dislocation. If the ankle injury is associated with lacerations of the skin in the area of the ankle joint, the injury is likely an open injury; **tetanus prophylaxis and antibiotic coverage** of skin flora should be given.

DIAGNOSTIC IMAGING

- Obtain radiographs of the patient's ankle, choosing the type of radiograph that may be performed and evaluated in the shortest duration of time.
- **Prereduction films** are often a valuable source of information; however, if significant neurovascular compromise is present and radiography would delay the time until reduction can be attempted, **prereduction films do not have to be obtained.**
- **Anteroposterior (AP) and lateral views** of the ankle are the most common and efficient means of radiographic assessment.
- *Computed tomography (CT) of the ankle, though superior to flat-plate radiography in revealing small fracture fragments, is usually not the test of choice, because it cannot be performed portably and may delay the time to reduction.*
- This test should be considered **only if neurovascular compromise is not present and the scan can be quickly performed and evaluated.**

- **Anesthesia**

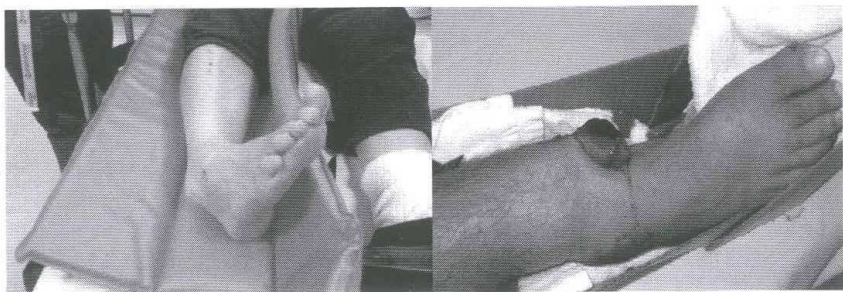
- Anesthesia for reduction of an ankle fracture or dislocation is usually performed by methods of **procedural sedation**, if the reduction is not taking place in the operating room under general anesthesia.

- **Positioning**

- Because of the application of procedural sedation, ankle reduction is usually performed with the patient in the **supine position** to provide immediate access to the patient's airway for bag-mask ventilation, if needed.
- The reduction is performed with the ipsilateral knee in a position of flexion, thus relieving tension on the Achilles tendon and making reduction easier.

- **Monitoring & Follow-up**

- Ortho referral (see indications below)
- If outpatient management is deemed appropriate, the patient should follow up in the next 2-3 days.
- Outpatient instructions should include the following:
 - **The patient should not bear weight on the affected ankle** until instructed otherwise upon follow-up with orthopedics; the ankle should remain in the splint at all times, and instructions as to the care of the splint must be given
 - **The patient should return for emergency care immediately** if pain increases, if the skin color of the distal foot changes, or if the injured leg exhibits any numbness, weakness, or change in temperature
 - **The patient should understand instructions for pain medicine** as deemed appropriate; narcotics, nonsteroidal anti-inflammatory drugs (NSAIDs), or both are usually warranted



APPROACH CONSIDERATIONS

- **Anterior dislocations of the talus** are associated with loss of a palpable **dorsalis pedis pulse** due to impingement from the displaced talus. This represents a vascular emergency, in that the true status of the artery cannot be accurately assessed while the ankle remains dislocated.
- If adequate reduction cannot be achieved, or if reduction has not restored the presence of a palpable pedal pulse, emergency operative management is indicated.
- *After each reduction attempt, **repeat the neurovascular examination** to ensure that blood flow has been maintained and no new sensory or motor compromise has occurred. If reduction has been achieved but neurovascular compromise is apparent after reduction, emergency operative management is indicated.*
- *If neurovascular compromise is present but reduction has not been achieved, operative management may be needed to reduce the injury, and limited future attempts should be made. If reduction cannot be accomplished after two or three attempts under optimal conditions, operative management should not be delayed further.*
- Once reduction is achieved and the neurovascular status of the limb is stable, **apply a long leg posterior splint with a sugar-tong component**, which immobilizes the joint in a position of 90° of flexion. All efforts should be made to avoid applying any material that may become constricting to the ankle; remarkable swelling may take place in the postreduction period.
- **The distal foot and toes should be left open to allow serial neurovascular checks.**
- **Repeat radiography** may now be performed to assess the adequacy of the reduction and document any associated fractures. Flat-plate radiography may consist of repeat anteroposterior and lateral views at a minimum; a mortise or additional view may be added to further describe the condition of the joint. Computed tomography (CT) of the ankle may provide additional information as to the presence of smaller fractures and the position of fracture fragments.

• COMPLICATIONS

- **Irreducible dislocation**
 - Osseous fragments, capsular ligaments, and ruptured tendons, as well as foreign bodies, may all become interposed in the anatomic joint space and make closed reduction impossible.
- Repeat forceful attempts at reduction can cause **additional soft-tissue injury** and **iatrogenic fractures** and can convert a closed injury into an open injury if the skin around the ankle is ruptured.

NEED FOR SURGICAL INTERVENTION

- Surgical intervention should be considered in the following scenarios:
 - *Failure to reduce the injury despite two or three attempts under optimal conditions*
 - *Increasing tension or tenting of the skin in a closed injury during reduction attempt*
 - *The presence of multiple other intra-articular fractures or subtalar dislocation demonstrated by radiography, in a neurovascularly intact injury*
 - *Amputation of the foot distal to the injury*
 - *Conversion of closed injury to open injury*
- *During closed reduction, if the skin over the ankle joint is ruptured (particularly over the malleoli), the injury has been converted into an open injury. **Tetanus prophylaxis** and **antibiotic coverage** of skin flora should be administered.*
- *If necessary, the wound should be surgically debrided.*

III. KNEE RELOCATION

- Do not delay reduction in limbs with obvious vascular impairment.
- Only patients with good peripheral pulses should undergo prereduction radiographs.
- Reduction is straightforward and often easily accomplished in the ED.
- After adequate sedation, longitudinal traction will relocate the majority of knee dislocations.



Figure 6.15.1. Lateral knee dislocation (before reduction) and Lateral knee dislocation after reduction.

- **Posterolateral dislocations** are particularly difficult and often require operative reduction.
- This is especially true when the medial femoral condyle button-holes through the medial aspect of the joint capsule and/or MCL — an occurrence that is often accompanied by a "dimple sign" overlying the medial aspect of the knee.

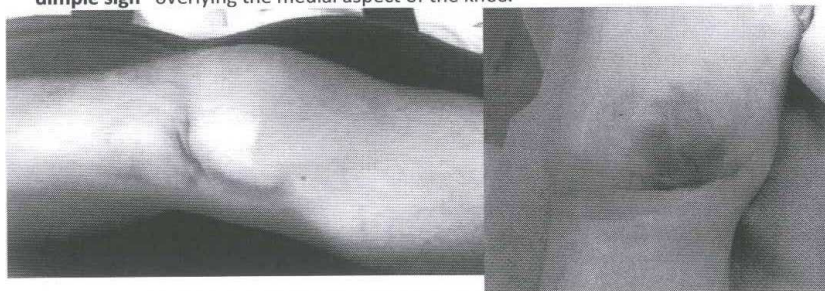


Figure 6.15.2. Dimple sign (medial aspect): Posterolateral Knee dislocation

- After reduction, splint the lower extremity in approximately 20 degrees of flexion to avoid postreduction re-dislocation, apply ice, and keep the knee elevated.
- **Postreduction radiographs** should be obtained, preferably before further ligamentous stressing/assessment.
- Postreduction hard signs of arterial injury should prompt emergent vascular surgical intervention that should not be delayed for arteriography.
- In this setting, **arteriograms** may indeed be contributory to the surgical decision matrix but can be performed in the operating room by the vascular surgeon with less contrast administration than traditional arteriography tends to use.
- All reduced knee dislocations without hard signs of arterial injury should be assessed with **ABI/API measurements**. Any reading of **less than 0.90** should prompt further imaging (ie, **arteriography vs CT angiography vs duplex sonography**), which should be decided upon in conjunction with the vascular consult.
- *All knee dislocations, regardless of emergent revascularization needs, should be admitted for serial perfusion checks.*

IV. HIP DISLOCATION AND REDUCTION

CLASSIFICATION

- **Simple:** pure dislocation without associated fracture
- **Complex:** dislocation associated with fracture of acetabulum or proximal femur

ANATOMIC CLASSIFICATION

- **Posterior hip dislocations**
 - The affected limb is shortened, adducted, and internally rotated, with the hip and knee held in slight flexion.
 - Patient may be unable to walk or adduct the leg.
 - Associated with:
 - *Osteonecrosis*
 - *Posterior wall acetabular fracture*
 - *Femoral head fractures*
 - *Sciatic nerve injuries*
 - *Ipsilateral knee injuries (up to 25%)*
- **Anterior hip dislocation**
 - The leg is externally rotated, abducted, and extended at the hip.
 - The femoral head may be palpated anterior to the pelvis.
 - Associated with:
 - *Femoral nerve injury*
 - *Femoral artery injury*
- **Central dislocation**
 - The leg is shortened, abducted or adducted, and internally or externally rotated, depending on the type and extent of penetration into the pelvis.
 - The typical posture of the leg with anterior or posterior hip dislocation may not be seen if an **associated femoral shaft fracture** is present.
 - The leg distal to the fracture assumes a neutral position, masking the usual rotation seen with a dislocation.
 - The incidence of missed hip dislocation is increased in the presence of a femoral shaft fracture.



Figure 6.15.3. Right ant. and left post. hip dislocations with acetabular fracture & Left Central hip dislocation

Presentation

- **Symptoms**
 - Acute pain, inability to bear weight, deformity
- **Physical Exam**
 - **ATLS:** 95% of dislocations with associated injuries
 - **Posterior dislocation (90%)**
 - Hip and leg in slight flexion, adduction, and internal rotation
 - Detailed neurovascular exam (10-20% sciatic nerve injury)
 - Examine knee for associated injury or instability
 - chest X-ray ATLS workup for aortic injury
 - **Anterior dislocation**
 - Hip and leg in flexion, abduction, and external rotation

Imaging

- **Radiographs**
 - Can typically see posterior dislocation on AP pelvis
 - Femoral head smaller than contralateral side
 - Shenton's line broken
 - Lesser trochanter shadow reveals internally rotated limb as compared to contralateral side
 - Scrutinize femoral neck to rule out fracture prior to attempting closed reduction
- **CT**
 - Helps to determine direction of dislocation, loose bodies, and associated fractures
 - Anterior dislocation
 - Posterior dislocation
 - Post reduction CT must be performed for all traumatic hip dislocations to look for
 - Femoral head fractures
 - Loose bodies
 - Acetabular fractures
- **MRI**
 - Controversial and routine use is not currently supported
 - Useful to evaluate labrum cartilage and femoral head vascularity

TREATMENT

- **NONOPERATIVE**
 - **Emergent closed reduction within 6 hours**
 - Indications: Acute anterior and posterior dislocations
 - Contraindications: Ipsilateral displaced or non-displaced femoral neck fracture

HIP REDUCTION TECHNIQUES

- There is a **6-hour window** for doing the reduction. If a neurovascular deficit is present the reduction should be **done sooner**. Closed reductions should initially be attempted.
- It is first necessary to give the patient **conscious sedation** which consists of an **IV Analgesia and muscle relaxant**.
- Several techniques can be tried:
 - **For Posterior hip dislocations:**
 - *Allis maneuver*
 - *Stimson maneuver*.
 - **For both Posterior and Anterior dislocations:**
 - *Reverse Bigelow maneuver*
 - *Leg-crossing maneuver*
 - *Longitudinal traction*
 - *Whistler maneuver*.
- **PRE- REDUCTION MANAGEMENT**
 - Perform with patient supine and apply traction in line with deformity regardless of direction of dislocation
 - Must have **adequate sedation and muscular relaxation** to perform reduction
- **POST- REDUCTION MANAGEMENT**
 - Assess hip stability after reduction
 - **Post reduction CT scan** required to rule out
 - Femoral head fractures
 - Intra-articular loose bodies/incarcerated fragments: may be present even with concentric reduction on plain films
 - Acetabular fractures
 - For simple dislocation, follow with **protected weight bearing for 4-6 weeks**

COMPLICATIONS

- **Femoral head osteonecrosis**
 - 5-40% incidence
 - Increased risk with increased time to reduction
- **Femoral nerve injury associated with femoral nerve injury**
- **Sciatic nerve injury**
 - 8-20% incidence
 - Associated with longer time to reduction
- **Post-traumatic arthritis**
 - Up to 20% for simple dislocation, markedly increased for complex dislocation
- **Recurrent dislocations**
 - Less than 2%
- **Complications of immobilization:**
 - *DVT*
 - *Pulmonary embolus*
 - *Pneumonia*

CHAPTER 16. JOINT ASPIRATION**I. KNEE ARTHROCENTESIS****• INDICATIONS**

- Indications for **diagnostic knee arthrocentesis** include the following:
 - Evaluation of monoarticular arthritis
 - Evaluation of suspected **septic arthritis**
 - Evaluation of joint effusion
 - Identification of intra-articular fracture
 - Identification of crystal arthropathy
- Indications for **therapeutic knee arthrocentesis** include the following:
 - Relief of pain by aspirating effusion or blood
 - Injection of medications (eg, corticosteroids, antibiotics, or anesthetics)
 - Drainage of septic effusion

• CONTRAINDICATIONS

- There are **no absolute contraindications** for knee arthrocentesis.
- Relative contraindications include the following:
 - **Cellulitis overlying the joint** - If arthrocentesis is performed, the patient should be admitted for the administration of intravenous (IV) antibiotics, even if the synovial fluid is not suggestive of infectious arthritis
 - **Skin lesion or dermatitis** overlying the joint
 - **Known bacteremia**
 - **Adjacent osteomyelitis**
 - **Uncontrolled coagulopathy**
 - **Joint prosthesis** - Preferably, a joint prosthesis is tapped by an orthopedist

• PERIPROCEDURAL CARE**• Equipment**

- The materials required for knee arthrocentesis include the following:
 - Sterile gloves and drapes / Gauze pads (5), 4 × 4 in, Skin preparatory solution
 - Lidocaine 1%, Syringes, 5 mL, 20 mL, 30 mL, 60 mL
 - Needles, 18 or 20 gauge and 25 or 27 gauge
 - Patients who are morbidly obese might require a 21-gauge spinal needle for arthrocentesis, Hemostat, Specimen tubes, Bandage

PATIENT PREPARATION**• Anesthesia**

- Patients who are anxious, in severe pain, or unable to cooperate with the procedure might require **procedural sedation and/or analgesia**.
- Local anesthesia is always warranted. After skin preparation, draping, and identification of the needle insertion site, use a 25- or 27-gauge needle to inject 2-5 mL of local anesthetic (eg, lidocaine 1%) into the subcutaneous tissue.

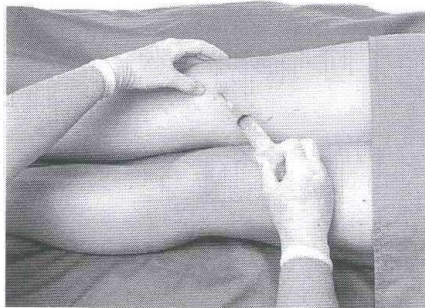
• Positioning

- **After obtaining informed consent**, place the patient **supine on a gurney**.
- Place a rolled towel below the patient's knee.

• Procedure Description

- The knee should be **fully extended** or just **slightly bent up to 15°**.
- The needle is held perpendicular to the leg and inserted **medially beneath the patella at approximately the 2-o'clock to 3-o'clock position**.

- The needle is inserted just posterior to the medial portion of the patella and is directed slightly posteriorly and slightly inferiorly.
- A lateral approach is also used in some cases (9-o'clock to 10-o'clock position).
- The prepatellar pouch can be emptied by gently applying pressure and squeezing the soft tissues, starting from the mid thigh and moving the hand towards the patella in order to shift the fluid toward the aspirating needle.

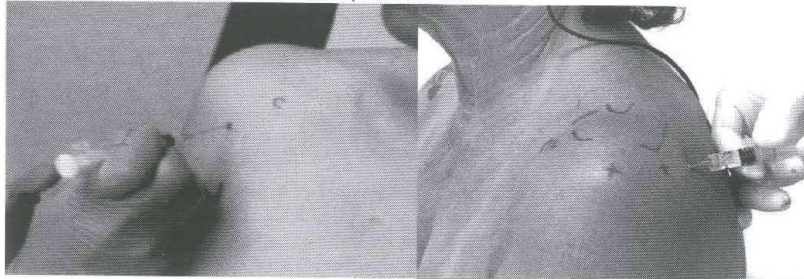


• COMPLICATIONS

- **Improper needle placement,**
- **Dry tap.**
- **Potential damage to cartilage**
- **Risk of introducing infection:** When arthrocentesis is performed through infected skin for the diagnosis of a potentially septic joint, intravenous antibiotics should be administered immediately after the procedure, and the patient should be admitted for continuation of the antibiotics.
- *Please do not send aspirations from joint Haemarthroses for cytology or culture when the precipitating event was trauma. These investigations should be reserved for spontaneous effusions or conditions such as crystal or septic arthritis.*
- **Full aseptic technique in all cases.**

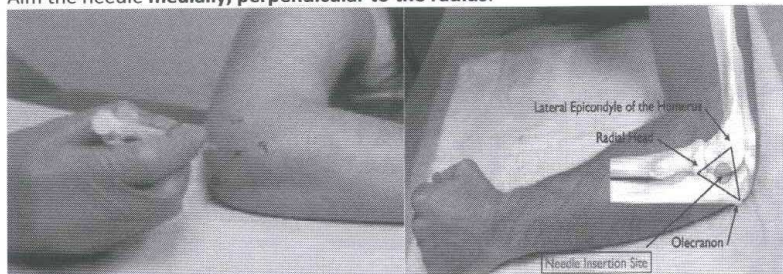
II. SHOULDER ARTHROCENTESIS

- **Anterior** – Just *lateral to the coracoid*, directing the needle *posteriorly*
- **Posterior** – Insert the needle **1 cm inferior and medial to the posterolateral corner of the acromium**. Direct the needle anteriorly and medially towards the coracoid. The glenohumeral joint is about 3-4 cm deep to the skin.



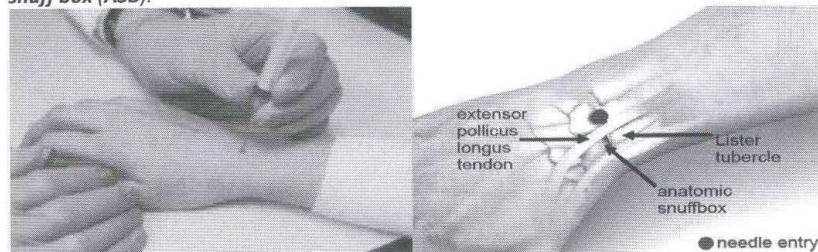
III. ELBOW ARTHROCENTESIS

- Place the elbow at **90 degrees of flexion, pronated**.
- Locate the **radial head**, **lateral epicondyle**, and lateral aspect of the **olecranon tip**.
- The centre of the **aconeus triangle** they form is the site of needle entry.
- Aim the needle **medially, perpendicular to the radius**.



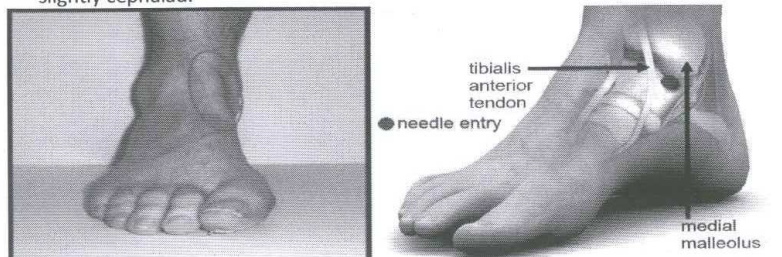
IV. WRIST ARTHROCENTESIS

- The wrist is held in a straight line with the forearm.
- A dimple is palpated dorsally over the radiocarpal joint, which provides the entry point for the needle. *The needle is held **perpendicular to the forearm** and **inserted dorsally**. Enter between **EPL** and **common extensor tendons**, ulnar to the **radial tubercle** and **anatomical snuff box (ASB)**.*



V. ANKLE ARTHROCENTESIS

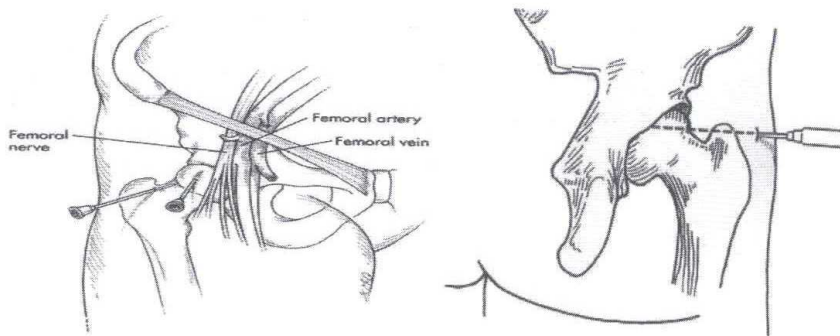
- **Subtalar** – Enter the joint just **below the lateral malleolus**
- **Tibiotalar** – Locate the sulcus between the medial malleolus and tibialis anterior / EHL tendons ... with the foot in neutral. Next plantar flex the foot and enter the sulcus, aiming slightly cephalad.



V. HIP JOINT ASPIRATION

• ANTERIOR APPROACH (Under Fluoroscopic guidance)

- **Femoral artery** may be palpated in femoral triangle, and may be used as a guide in aspirating the hip joint;
- Palpate the femoral pulse just as it exits the inguinal ligament;
- Entry point is 2cm lateral to the artery (at the inguinal ligament) and preferably by at least 2 cm below the inguinal ligament; Needle entry is then straight down into the lateral half of the joint cavity.
- **Disadvantages:** if the surgeon is not in the capsule when the contrast dye is injected, then contrast material will Collect and will obstruct needle visualization.



• LATERAL APPROACH

- Greater trochanter is palpated & needle inserted just **anterior to its superior tip**; Needle is directed 45°cephalad, & parallel to table (patient is supine); Femoral neck will usually be met & needle can then be directed slightly cephalad and proximal to enter the hip joint;
- Greater trochanter is palpated, & **needle is inserted from side**, in front of its upper margin and approx parallel to femoral neck, so that needle enters capsule obliquely after passing thru attachments of **gluteus medius & minimus**;
- **Disadvantages:** in patients with large thighs, the needle may not be long enough to reach the joint;

• MEDIAL APPROACH

- Needle is inserted just **posterior to the insertion of the adductor longus muscle**, and **anterior to the gracilis**;
- Fluoroscopy is then used to direct the needle into the hip joint
- If the patient has a hip prosthesis, a skin entry point medial or lateral to the prosthesis avoids overlap that prohibits visualization of the needle fluoroscopically.
- US has been increasingly used to access the hip joint and provides the advantage of avoiding radiation exposure. It is particularly useful for aspiration of the hip in children.

CHAPTER 17. ASSESSMENT OF TRAUMA PATIENT**I. PRIMARY SURVEY****Case Presentation**

- Your trauma patient from recent ambulance call has arrived... A trauma call was activated and the team assembled. The patient was transferred onto a bed in the trauma bay, and removed from a spinal board used for transfer. Handover and vital signs are being obtained as the trauma team get to work. As the **Trauma Team Leader**, you're going to need to know your stuff to be able to coordinate the team's initial assessment and management of this trauma patient.

**Q1. What are the 5 key components of the primary survey in major trauma?**

- ATLS (8th edition) emphasizes the **ABCDE** approach:
 - Airway maintenance with cervical spine protection
 - Breathing and ventilation
 - Circulation with haemorrhage control
 - Disability (neurological status)
 - Exposure and environmental control (completely undress but avoid hypothermia)
- This assessment can usually be performed in less than 10 seconds.
- **Handy tips:**
 - Talk to the patient. Any verbal response strongly suggests that for the moment at least their ABCDE's are okay!
 - Once identified, deal with a problem BEFORE moving on in your assessment ("**FIND the bleeding, STOP the bleeding**").
 - After any intervention, return to the start of the primary survey.

Q2. What does AIRWAY MAINTENANCE WITH CERVICAL SPINE CONTROL involve?

- Airway assessment and maintenance:
 - **Ensure patency** and consider if airway protection is required.
 - Escalate **from simple to advanced** techniques as required.
 - **Suction and Magill's forceps** should be immediately available.
 - **Employ simple airway manoeuvres** – jaw thrust and chin lift. Head tilt inappropriate with cervical spine control.
 - **Use airway adjuncts** – Oropharyngeal airway usually only a temporizing measure if tolerated as usually requires definitive airway. Nasopharyngeal airways are inappropriate in head and facial trauma due to risk of intracranial passage.
- Consider the **need for a definitive airway**, which may be required for A, B C or D reasons:
 - **A** – eg: **impending airway obstruction** (burns, penetrating or blunt neck injury) or injury that may distort airway anatomy (e.g. neck haematoma)
 - **B** – eg: **Respiratory insufficiency** due to a large pulmonary contusion, flail chest, or other thoracic injury.

- C – eg: **Multisystem trauma** with shock
- D – eg: **Reduced GCS** (especially <8), penetrating cranial vault injury
- Intubation may also be advisable prior to invasive procedures (e.g. chest tube insertion) or patient transfer. All ED intubations should be regarded as difficult airways and every operator should have a 'plan B' prior to commencing.
- Surgical airways (e.g. cricothyrotomy) are required in the '**can't intubate, can't ventilate scenario**'.
- Patients that require urgent, but not emergent intubation (e.g. facial burns) may be best intubated by awake fiberoptic intubation with anesthesia and ENT in attendance.
- **Major trauma patients should be suspected of having cervical spine injuries.** Until the cervical spine is cleared, spinal precautions should be employed.
 - *Control cervical spine with a hard collar, sandbags and tape*
 - *Easy access to airway may mandate manual cervical spine immobilization by a third party until airway control is achieved*

Q3. What does **ASSESSMENT AND MANAGEMENT OF BREATHING AND VENTILATION** involve?

- **Assessment**
 - **Respiratory Rate and SpO₂**
 - **Inspection:** external signs of trauma, asymmetrical chest movements
 - **Palpation:** may reveal unsuspected injury e.g. crepitus / surgical emphysema.
 - **Percussion** – often difficult in a noisy trauma bay
 - **Auscultation** – listening for air entry bilaterally, gauge adequacy and added sounds
 - **Trachea** – palpate to see if deviated, although true tracheal deviation due to a tension pneumothorax is pre-terminal and it is unlikely to be the only sign
 - May be appropriate to log roll at this stage if concerned about a posterior chest injury.
- **Management**
 - **High flow oxygen 15L/min** via non-rebreather mask on arrival
 - Non-invasive ventilation is rarely indicated in trauma patients
 - Patients requiring respiratory support are usually intubated and mechanically ventilated
 - **Needle thoracotomy, finger thoracotomy or Intercostal Catheter Drain** may be required urgently

Q4. What does **ASSESSMENT AND MANAGEMENT OF CIRCULATION** with haemorrhage control involve?

- **Assessment**
 - **Pulse Rate, Blood Pressure, Capillary Refill** and the **Warmth of Peripheries**
 - Systematically look for **evidence of bleeding** (external and internal).
 - The key areas are: "**Blood on the floor and 4 more**" = intrathoracic, intraperitoneal, retroperitoneal, pelvis/thigh.
 - Removal of all prehospital bandaging is vital – a poorly applied 'turban' can mask major scalp haemorrhage.
- **Management**
 - **Insert 2 large bore** (at least 16 gauge) intravenous cannulae, ideally in the antecubital fossae. If this cannot be rapidly achieved obtain **intraosseous access**.
 - **Urgent investigations:** Crossmatch blood, Venous blood gas (Lactate and Hb).
 - Others tests include full blood count, urea and electrolytes, creatinine, glucose, coagulation profile and lipase. These rarely alter initial management.

- **IV fluids** — Normal Saline or Hartman's Solution — 1-2 L STAT. Change to **blood** if remains haemodynamically unstable after 2 L of crystalloid, or earlier if obvious signs of major bleeding.
- This approach is being superseded by the concept of **damage control resuscitation**.
- **Haemorrhage control:**
 - Most external bleeding can be at least temporarily controlled with **direct pressure**, **tourniquets** or by **tying off vessels**.
 - Other measures are considered in 'major haemorrhage', and ultimately damage control surgery may be needed.

Q5. What does DISABILITY (neurological evaluation) involve?

- **Assessment**
 - Assess **GCS** and document it's components (e.g. E4, V5, M6 = GCS 15)
 - Assess **pupillary size and responsiveness** (if you can open the eyelids due to swelling, consider using ocular ultrasound)
 - Assess **gross motor and sensory function** in all 4 limbs
 - If you suspect a spinal injury is present a full neurological assessment is vital at the earliest opportunity — check for **priapism**, **loss of anal sphincter tone** and the **bulbocavernosus reflex**
 - **Check glucose**
- **Management**
 - **Airway maintenance** (see above)
 - **Seizure control** — **Midazolam 5-10mg IV**, followed by **Phenytoin 18mg/kg IV** over 30 minutes.
 - **Treat hypoglycemia** (glucose <3 mmol/L) with 50 mL 50% glucose
 - **Anxiety or agitation** — treat pain, shock and search for underlying cause
 - **Treat raised intracranial pressure:**
 - *30 degree head up positioning,*
 - *Analgesia and sedation,*
 - *Neuromuscular blockade,*
 - *Manitol or hypertonic saline,*
 - *Arrange for urgent surgical decompression*

Q6. What does EXPOSURE AND ENVIRONMENTAL CONTROL involve?

- While maintaining thermostasis, **completely expose the patient**
- If not yet done, consider **log-rolling** the patient now
- Areas where potentially life threatening injuries can be missed are:
 - Back of head, Back, Buttocks, Perineum, Axillae and Skin folds

Q7. What should be EXAMINED FOR IN THE NECK of a trauma patient?

- Look for **TWELVE** things (OK, there's only six, so check them twice...):
 - Tracheal deviation
 - **Wounds**
 - External markings
 - Laryngeal disruption
 - Venous distention
 - Emphysema (surgical)
- These findings suggest life-threatening injuries to the neck or thorax (e.g. tension pneumothorax, cardiac tamponade).

- Also, don't forget to check for a **Horner's syndrome** in possible neck trauma!
- A widely-used mnemonic for the **6 killer conditions** to think of, and actively search for, during the primary survey is "**ATOM-FC**":
 - *Airway obstruction or disruption*
 - *Tension pneumothorax*
 - *Open pneumothorax*
 - *Massive haemothorax*
 - *Flail chest*
 - *Cardiac tamponade*

II. SECONDARY SURVEY

- A focused history and physical exam should be performed after the initial assessment.
- It is assumed that the life-threatening problems have been found and corrected.
- If you have a patient with a life-threatening problem that requires intervention (i.e. CPR) you may not get to this component. The main purpose of the focused history and physical is to discover and care for a patient's specific injuries or medical problems.

1. FOCUSED HISTORY AND PHYSICAL EXAM

- The focused history and physical exam includes a physical examination that focuses on a specific injury or medical complaint, or it may be a rapid examination of the entire body.
- It also includes obtaining a patient history and vital signs.
- **Patient History**
 - **S** - Signs/symptoms
 - **A** - Allergies
 - **M** - Medications
 - **P** - Past medical history
 - **L** - Last oral intake/ LMP
 - **E** - Events leading to the illness or injury
- **Rapid assessment**
 - This is a quick, less detailed head - to toe assessment of the most critical patients
- **Focused assessment**
 - This is an exam conducted on stable patients. It focuses on a specific injury or medical complaint.
- **Vital signs**
 - This include: BP, HR, RR, SpO2, skin signs, pupils.
 - **Pulse** - Assess for rate, rhythm, and strength
 - **Respiration** - Assess for rate, depth, sound, and ease of breathing
 - **Skin signs** - Assess for color, temperature, and moisture
 - **Pupils** - Check pupils for size, equality, and reaction to light. Constricted pupils in a mass casualty event are highly suggestive of nerve agent/organophosphate toxicity.

2. HEAD TO TOE EXAMINATION OF A TRAUMA PATIENT

- The physical examination of the patient should take no more than two to three minutes
- **Head**
 - Check the scalp for cuts, bruises, swellings, and other signs of injury.
 - Examine the skull for deformities, depressions, and other signs of injury.
 - Inspect the eyelids/eyes for impaled objects or other injury.
 - Determine pupil size, equality, and reactions to light.
 - Note the color of the inner surface of the eyelids.

- Look for blood, clear fluids, or bloody fluids in the nose and ears.
- Examine the mouth for airway obstructions, blood, and any odd odors.
- **Neck**
 - Examine the patient for point tenderness or deformity of the cervical spine.
 - Any tenderness or deformity should be an indication of a possible spine injury.
 - If the patient's C-spine has not been immobilized immobilize now prior to moving on with the rest of the exam.
 - Check to see if the patient is a neck breather, check for tracheal deviation
- **Chest**
 - Examine the chest for cuts, bruises, penetrations, and impaled objects.
 - Check for fractures.
 - Note chest movements a look for equal expansion.
- **Abdomen**
 - Examine the abdomen for cuts bruises, penetrations, and impaled objects.
 - Feel the abdomen for tenderness.
 - Gently press on the abdomen with the palm side of the fingers, noting any areas that are rigid, swollen, or painful.
 - Note if the pain is in one spot or generalized.
 - Check by quadrants and document any problems in a specific quadrant.
- **Lower Back**
 - Feel for point tenderness, deformity, and other signs of injury
- **Pelvis**
 - Feel the pelvis for injuries and possible fractures.
 - After checking the lower back, slide your hands from the small of the back to the lateral wings of the pelvis.
 - Press in and down at the same time noting the presence of pain and/ or deformity
- **Genital Region**
 - Look for wetness caused by incontinence or bleeding or impaled objects.
 - In male patients check for priapism (persistent erection of the penis). This is an important indication of spinal injury.
- **Lower Extremities**
 - Examine for deformities, swellings, bleedings, discolorations, bone protrusions and obvious fractures.
 - Check for a distal pulse. The most useful is **the posterior tibial pulse** which is felt behind the medial ankle.
 - If a patient is wearing boots and has indications of a crush injury do not remove them.
 - Check the feet for motor function and sensation.
- **Upper Extremities**
 - Examine for deformities, swellings, bleedings, discolorations, bone protrusions and obvious fractures and Check for the **radial pulse** (wrist).
 - In children check for capillary refill. Check for motor function and strength.

III. TERTIARY SURVEY

- The tertiary survey is a repetition of the secondary survey that again aims to pick up 'missed' injuries. This may occur on multiple occasions over the days following injury.
- The multiple injured motorcyclist's broken pinkie can be easy to miss during initial assessment!

CHAPTER 18. MEDICALLY UNWELL PATIENTS

I. PRIMARY SURVEY

• COMMON EMERGENCY PRESENTATIONS

- Coma
- Difficulty in breathing
- Chest pain
- Collapse with hypotension
- Gastrointestinal bleeding
- Collapse with altered consciousness
- Abdominal pain
- Headache
- Seizures

RAPID PRIMARY SURVEY

• AIRWAY ASSESSMENT

- Is there **evidence of airway obstruction** (noisy breathing, stridor, obstructive respiratory pattern)?
- Is there **failure of airway protection** (pooling of secretions, absence of spontaneous swallowing)?
- Is there **evidence of mucosal oedema** (anaphylaxis) or **foreign body aspiration**?
- Assess the airway looking for signs of obstruction and to check that the patient is maintaining and protecting the airway.
- The unconscious patient is at significant risk of *passive regurgitation* and pulmonary aspiration even if the airway is maintained with simple techniques and positioning.
- Failure to clear blood, saliva, or mucus from the oropharynx and absence of spontaneous swallowing indicate a **failure of airway protection**.
- Although the full range of basic and advanced airway management interventions should be available to manage such patients, simple adjuncts (especially nasopharyngeal airways), postural drainage, and head and neck positioning may be sufficient.

• BREATHING ASSESSMENT

- Is there evidence of an **increased work of breathing** (tachypnoea, accessory muscle use, recession)?
- Is there evidence of **hypoxia or fatigue** (cyanosis, feeble respiratory effort)?
- Is there evidence of pneumothorax, asthma, anaphylaxis, heart failure, pneumonia, or chronic obstructive pulmonary disease?
- To assess breathing, look for signs of increased respiratory effort, inadequate ventilation, and common physical signs associated with respiratory and cardiovascular disease.
- *An increased RR, use of accessory muscles, splinting of the diaphragm, and recession of the chest wall are sensitive indicators of an increased work of breathing.*
- Tachypnoea alone may reflect a very wide range of disease processes and it should not be assumed to reflect a breathing problem in the absence of other signs of respiratory distress. If wheeze is present, decide if the sound occurs mainly during inspiration (*stridor*) or expiration (most likely to be attributable to lower airways obstruction)

• CIRCULATION ASSESSMENT

- Is there **evidence of bleeding** (haematemesis, malaena, concealed bleeding)?

- Is there **evidence of shock** (tachycardia, prolonged capillary refill time, increased respiratory rate, low blood pressure)?
- Does the patient have **evidence of sepsis** (any two of heart rate >90, respiratory rate >20 and temperature >38°C or <36°C)?
- Is there evidence of **acute coronary syndrome, heart failure, or arrhythmias**?
- Assessment of the circulation should identify the presence of shock and a systemic inflammatory response to infection.
- Acute gastrointestinal haemorrhage may be missed if the clinical signs of bleeding are not assessed.
- Finally, assessment of the circulation in medical emergencies includes an assessment of heart rhythm and a search for evidence of heart failure and myocardial dysfunction (*tachycardia, 3rd or 4th heart sounds, systolic murmur*).
- **DISABILITY ASSESSMENT**
 - Is there an **altered level of consciousness**?
 - Is the patient **fitting**?
 - Is the patient **hypoglycaemic**?
 - Is there any **evidence of meningism** (neck stiffness, photophobia)?
 - Are there any **localising signs** (pupils, cranial nerves, limbs)?
- Assessment of disability entails a mini-neurological examination starting with level of consciousness, mental state, pupil signs, localising signs, posture, and limb function.
- **EXPOSURE ASSESSMENT**
 - Is there a **rash** (urticaria, purpura)?
 - Is the patient **hypothermic or feverish**?
 - Are there any obvious **physical stigmata of chronic disease**?
- The patient should also be exposed as much as practicable to look for evidence of a rash (urticaria or purpura), jaundice, anaemia, pitting oedema, and physical manifestations of chronic disease.
- An accurate assessment of temperature is essential in assessing whether the patient is feverish or hypothermic.

II. SECONDARY SURVEY

FOCUSED HISTORY AND PHYSICAL EXAM

- The focused history and physical exam includes a physical examination that focuses on a specific medical complaint, or it may be a rapid examination of the entire body.
- It also includes obtaining a patient history and vital signs.
- **Patient History**
 - **S** - Signs/symptoms
 - **A** - Allergies
 - **M** - Medications
 - **P** - Past medical history
 - **L** - Last oral intake/ LMP
 - **E** - Events leading to the illness
- **Rapid assessment**
 - This a quick, less detailed head - to toe assessment of the most critical patients
- **Focused assessment**
 - This is an exam conducted on stable patients. It focuses on a specific medical complaint.

- **Vital signs**

- This include: BP, HR, RR, SpO₂, skin signs, pupils.
- **Pulse** - Assess for rate, rhythm, and strength
- **Respiration** - Assess for rate, depth, sound, and ease of breathing
- **Skin signs** - Assess for color, temperature, and moisture
- **Pupils** - Check pupils for size, equality, and reaction to light. Constricted pupils in a mass casualty event are highly suggestive of nerve agent/organophosphate toxicity.

- **In an emergency situation pay particular attention to following signs and symptoms:**

- **Head**

- Is headache present
- Are the pupils are the pinpoint, dilated, asymmetrical in size
- Are the conjunctiva injected, draining,
- Does the patient complain of eye pain, photophobia or blurring of vision
- Is salivation, drooling, and/or rhinorrhea present
- Is nasal flaring present
- Note skin color - i.e. is the patient cyanotic
- Note the smell of the patients breath
- Is the patients throat sore, red

- **Neck**

- Is stridor present
- Are the muscles in the neck "pulling"

- **Chest/Lungs**

- Note the presence of increased work of breathing i.e. retractions, increased rate
- Note the presence of stridor
- Note the presence of wheezing, rhonchi, rales, decreased breath sounds
- Note the presence of central cyanosis
- Does the patient complain of burning in the chest or chest pain

- **Heart/Circulation**

- Note the presence of irregular, fast or slow heart rhythms
- Note the presence of diminished or absent peripheral pulse
- Note the presence of prolonged capillary refill in children
- Note the color and temperature of the distal extremities

- **Abdomen**

- Is the abdomen painful, tense, distended or rigid?
- Does the patient have cramping, vomiting or diarrhea

- **Pelvis**

- Check for incontinence of urine or feces

- **Neurological**

- What is the patient's mental status? Is he (she) seizing?
- Is the patient dizzy?
- Did syncope occur?
- Was there sudden collapse
- Does he (she) have muscle twitching?

- **Skin**

- Is the skin painful, burning numb or tingly
- Is the skin erythematous
- Are there vesicles, bullae
- Is there necrosis

CHAPTER 19. MECHANICAL VENTILATOR**I. NON-INVASIVE VENTILATION (NIV)**

- Include machines used to ventilate and oxygenate patients without the need to perform the invasive procedure of intubation.
- *These machines can only be used on a spontaneously breathing patient.*
- Another acronym commonly used to describe NIPPV is **NIV**, which essentially stands for **Non-Invasive Ventilation**.
- The two most common forms of NIV are:
 - CPAP
 - BiPAP

I. CPAP

- This is **Continuous Positive Airway Pressure**.
- It's a pressure exhale applied during the respiratory cycle that helps keep air passages open so that the next breath comes in easier.
- Since it keeps the airways patent, it assures **adequate oxygenation** and is often prescribed to increase oxygenation.

Indications	Hypoxemia due to: <ul style="list-style-type: none"> ◦ CCF, CPO, Pneumonia, asthma, COPD, ◦ Near drowning ◦ Obstructive Sleep Apnea
Cautions	<ul style="list-style-type: none"> ◦ Cardiogenic shock ◦ Agitated patient
"CARS"	<ul style="list-style-type: none"> ◦ Right ventricular failure ◦ Severe obstructive airways disease
Contraindications	<ul style="list-style-type: none"> ◦ Immediate endotracheal intubation indicated ◦ Respiratory arrest or inadequate spontaneous ventilation ◦ Worsening life threatening hypoxia ◦ Unconscious patient unable to protect own airway
How to deliver NIV	<ul style="list-style-type: none"> ◦ Correctly fitting mask ◦ Supplemental O₂ ◦ Commence PEEP at 5-7.5 cm H₂O and increase to 10cm as tolerated ◦ Continue for 30min/hr until reduction in dyspnoea and saturations are maintained off NIV
Complications	<ul style="list-style-type: none"> ◦ Dry mouth ◦ Aspiration ◦ Worsening right ventricular failure
"DAW - HIPS"	<ul style="list-style-type: none"> ◦ Hypercapnoea, ◦ Intolerance due to anxiety, ◦ Pneumothorax ◦ Skin/Eye discomfort,

II. BiPAP

- This is an acronym for **Bi-level** (or **Biphasic**) **Positive Airway Pressure**.
- It provides a combination of both **IPAP** and **EPAP**.
- **Indications of NIV:**
 - **Hypercapnic respiratory failure during an acute exacerbation of COPD with:**
 - *Arterial pH <7.35.*
 - *Arterial PaCO₂ >6kPa (if acute onset).*
 - *Tachypnoea >25 breaths/min*

A. IPAP

- This is **Inspiratory Positive Airway Pressure**.
- It is a pressure during inspiration that assists a patient obtain an adequate **tidal volume**.
- Because it provides assistance with inhalation, it therefore **decreases the work of breathing required** to get air in.
- Because it assures **adequate ventilation**, it is often prescribed to **blow off carbon dioxide (CO₂)**.

B. EPAP

- This is **Expiratory Positive Airway Pressure**.
- It is the same thing as CPAP.
- EPAP is simply used here so you know your talking about CPAP on a BiPAP machine.
- EPAP is used to **improve oxygenation**.
- **INCLUSION CRITERIA FOR NIV**
 - Primary diagnosis of COPD exacerbation
 - Able to protect airway
 - Conscious and cooperative
 - Patients wishes considered
 - Potential for recovery of quality of life that will be acceptable to the patient
 - NIV can be considered in the unconscious if within a critical care setting or intubation is inappropriate
- **EXCLUSION CRITERIA FOR NIV ARE:**
 - Life threatening hypoxaemia
 - Intubation and ventilation is possible and would be in patients best interests
 - Inability to protect the airway
 - Confusion/agitation
 - Undrained pneumothorax
 - Fixed upper airway obstruction
 - Facial burns/trauma
 - Recent facial or upper airway surgery
 - Vomiting
 - Copious respiratory secretions
 - Bowel obstruction
 - Upper gastrointestinal surgery
 - Severe co-morbidity
 - Patient moribund

II. INVASIVE MECHANICAL VENTILATION

• INDICATIONS FOR INVASIVE MECHANICAL VENTILATION:

- A decision to intubate and proceed with mechanical ventilation should normally be made **within 4 hours of starting NIV**, as improvements should usually be apparent during this time.
- Patients with COPD should be considered for ITU treatment when necessary, especially if they are more unwell i.e. **pH < 7.26**.
- The Global Initiative for Chronic Obstructive Lung Disease 2013 guideline states the following may be **indications for invasive mechanical ventilation**:
 - NIV failure
 - Inability to tolerate NIV
 - Respiratory or cardiac arrest
 - Respiratory pauses with loss of consciousness or gasping for air
 - Reduced consciousness or uncontrolled agitation
 - Massive aspiration
 - Persistent inability to remove respiratory secretion
 - Heart rate <50 with loss of alertness
 - Haemodynamic instability unresponsive to fluid and vasopressors
 - Life threatening hypoxaemia

COMPLICATIONS OF MECHANICAL VENTILATION

• Complications of intubation

- *Upper airway and nasal trauma, Tooth avulsion,*
- *Oral-pharyngeal laceration,*
- *Laceration or hematoma of the vocal cords,*
- *Tracheal laceration,*
- *Perforation,*
- *Hypoxemia,*
- *Intubation of the esophagus.*
- *Inadvertent intubation of the right mainstem bronchus*
- *Aspiration rates are 8–19% in intubations performed in adults without anesthesia.*
- *Sinusitis, tracheal necrosis or stenosis, glottic edema, and ventilator-associated*
- *Pneumonia may occur with prolonged use of endotracheal tubes.*

• Ventilator-induced lung injury (VILI)

- *Barotrauma*
- *Volutrauma*

III. SETTING UP NIV

- Initial settings for ventilation may be summarized as follows:
 - **Assist-control mode**
 - **Tidal volume** set depending on lung status
 - *Normal = 12 mL/kg ideal body weight;*
 - *COPD = 10 mL/kg ideal body weight;*
 - *ARDS = 6-8 mL/kg ideal body weight*
 - **Rate** of 10-12 breaths per minute
 - **FIO₂ of 100%**
 - Sighs rarely needed
 - **PEEP** only as indicated after first arterial blood gas determination, ie, shunt greater than 25% and inability to oxygenate with an FIO₂ less than 60%
- Parameters commonly used to assess a patient's readiness to be weaned from mechanical ventilatory support include the following:
 - *Respiratory rate less than 25 breaths per minute*
 - *Tidal volume greater than 5 mL/kg*
 - *Vital capacity greater than 10 mL/kg*
 - *Minute ventilation less than 10 L/min*
 - *PaO₂/FIO₂ greater than 200*
 - *Shunt (Qs/Qt) less than 20%*
 - *Negative inspiratory force (NIF) less than (more negative) -25 cm water*
 - *F/Vt less than 105, or less than 130 in elderly patients*
- **INITIATING NIV**
 - Commence BiPAP at **IPAP 10cm H₂O / EPAP 4cm H₂O**.
 - **Increase FIO₂** to improve O₂ saturation to **>90%**.
 - **Repeat ABG after 1 hr** of NIV treatment.
 - **Titrate IPAP:** if pH<7.35, RR >25/min, PaCO₂>6 kPa or persistent use of accessory muscles.
 - **Titrate EPAP:** if persistent hypoxia.
 - *Titrate in increments of 2cm H₂O to peak IPAP 20 / EPAP 8.*
 - *Repeat ABG after 4 hrs of NIV; titrate pressures as above.*
 - *NIV should be used for a minimum of 16 hours / 24 hours initially, reducing to 12 hours on Day 2, and 8 hours on Day 3 as the clinical setting permits.*
- **FULL VENTILATION RECONSIDERED IF:**
 - Arterial pH<7.2.
 - Arterial pH 7.2 - 7.25 on two occasions 1 hr apart.
 - Hypercapnic coma **GCS** <8 and PaCO₂>8 kPa.
 - PaO₂<6 kPa despite maximum tolerated FIO₂.
 - Cardiorespiratory arrest.

SUMMARY OF INITIAL VENTILATOR SETUP

- Before commencing NIV there should be a clear plan of escalation and ceilings of treatment. This should be documented in the patient notes
- NIV should be clearly prescribed, a full facemask should be used for the first 24 hours, and an initial **IPAP of 10 cm H₂O and EPAP of 4-5 cm of water** should be used.

- This should be increased rapidly at a rate of approximately **5 cm of water every 10 minutes to a target of 20 cm H₂O**, or patient unable to tolerate further, or therapeutic response achieved.
- **Arterial blood gas (ABG)** analysis should be performed at **baseline, 1 hour after commencing NIV and 1 hour after changing settings**
- All patients should be on **continuous pulse oximetry and ECG monitoring for the first 12 hours**.

- **TREATMENT FAILURE**

- Is medical treatment optimal?
- Is physiotherapy needed (particularly for sputum retention)?
- What complications have developed (beware **PNEUMOTHORAX** or **ASPIRATION** etc.)
- Check the pressures actually being achieved (visible on the screen).

- **If PaCO₂ remains high**

- Too much O₂? Maintain SpO₂ between 85% to 90%
- Excessive mask leakage?
- Is circuit set up correctly?
- Is patient synchronising with ventilator – adjust breathing rate and/or inspiratory and/or expiratory trigger
- Is re-breathing occurring? - Check patency of expiratory valve (if fitted). Consider increasing EPAP
- Is ventilation adequate – ?increase IPAP (increments of 2cm H₂O to alleviate resp distress)

- **If PaCO₂ improves but PaO₂ remains low**

- Increase FiO₂
- Consider increasing EPAP by increments of 2cm H₂O.
- NB: **keep difference between IPAP and EPAP ≥ 6 cmH₂O** - so you may need to also increase IPAP.

- **INFECTION CONTROL**

- **Disposable masks** and exhalation ports should be disposed of.
- **Headgear should be washed** in a washing machine – be careful with the Velcro straps.
- **Use a bacterial filter** between the tube and the BiPaP machine to reduce contamination risk to machine.

CHAPTER 20. DRUG TO FACILITATE VENTILATION

• PHARMACOLOGICAL MANAGEMENT

- The class of medication used needs to match the underlying cause of discomfort.
- In a ventilated patient, this is often multifactorial, and thus a combination of pharmacotherapy may be required. When considering combinations of drugs, knowledge of their context sensitive half-times is essential.

Class of drug	Examples	Advantages	Disadvantages
I.V. induction agents	Propofol Also: Thiopental Etomidate Ketamine	Reduced duration of ventilation compared with benzodiazepines; Little significant accumulation	Bradycardia; Decreased systemic vascular resistance; Propofol infusion syndrome
Neuroleptic agents	Haloperidol; chlorpromazine	Neuroleptosis; Profound sedation; Minimal respiratory depression	Extrapyramidal signs; hypotension; Q-T prolongation
Benzodiazepines	Midazolam; Lorazepam; Diazepam	Anxiolysis; haemodynamic stability	Dependence; Withdrawal agitation; Active metabolites (midazolam, diazepam); Long elimination half-life (Diazepam)
Opioids	Morphine; Fentanyl; Alfentanil; Remifentanyl	Analgesia; Anxiolysis	Respiratory depression; Bradycardia; Hypotension; Nausea; Constipation
Alpha agonists	Clonidine	Analgesia; Anxiolysis; Minimal respiratory depression	Rebound hypertension; Hypotension; Bradycardia; Elimination delayed in renal failure

NEUROMUSCULAR BLOCKING AGENTS

- Neuromuscular blocking agents **do not provide sedation**, and are only occasionally used in critical care due to concerns about chronic muscle weakness and the risk of paralysis without adequate sedation.
- Development of myopathy is directly related to duration of infusion.
- **Indications include:**
 - Invasive ventilation modes (e.g. Inverse ratios, high pressures);
 - Control of ventilation in those with a high respiratory drive;
 - Reduction of oxygen consumption in critically hypoxaemic patients;
 - Control of raised intracranial pressure.

CHAPTER 21. PATIENT FIGHTING VENTILATOR**CAUSES OF RESPIRATORY DISTRESS IN MECHANICALLY VENTILATED PATIENTS**

Ventilator
<ul style="list-style-type: none"> ○ Inadequate ventilator settings ○ Ventilator circuit leak ○ Ventilator malfunction ○ Inadequate ventilation due to caudal or esophageal misplacement of the endotracheal tube
Airway (increased ΔP_{peak}-P_{plat})
Endotracheal tube
<ul style="list-style-type: none"> ○ Patient biting tube ○ Balloon cuff leak, deflation, or rupture ○ Increased airway resistance imposed by heat and moisture exchanger ○ Obstruction by secretions, blood, or foreign object
Bronchospasm
<ul style="list-style-type: none"> ○ Obstruction of lower airways by secretions, blood, or foreign object
Pulmonary parenchymal disease (ΔP_{peak}-P_{plat} unchanged or decreased)
<ul style="list-style-type: none"> ○ Pneumonia ○ Atelectasis ○ Pulmonary edema (cardiogenic or non-cardiogenic) ○ Aspiration of oropharyngeal or gastroesophageal contents ○ Pulmonary embolus (thromboembolism or gas embolism) ○ Migration of tube into right mainstem bronchus
Extrapulmonary causes
<ul style="list-style-type: none"> ○ Pneumothorax ○ Pleural effusion ○ Abdominal distension (eg, ascites) ○ Delirium, anxiety, pain, acute neurological event

P_{peak}: peak inspiratory pressure; P_{plat}: plateau pressure.

- **OVERVIEW**

- Patient-Ventilator Dyssynchrony occurs when the patient's demands are not met by the ventilator, resulting from problems with:
 - Timing of inspiration / Adequate inspiratory flow for demand
 - Timing of the switch to expiration / Duration of inspiration

- **VENTILATION STRATEGIES**

- **Total Ventilator-controlled Mechanical Support:**
 - Patients breathing pattern is totally controlled by ventilator (pressure generated by patient abolished by paralysis and sedation)
 - Risks: prolonged sedation and paralysis, respiratory muscle atrophy, overdistention, patient discomfort, prolonged weaning
- **Partial Patient-Controlled Mechanical Support:**
 - Spontaneously breathing activity preserved
 - Weaning accelerated, preservation of respiratory muscle power
 - Risks: requires synchronization of patients ventilatory demand and ventilator settings.

CAUSES

- **Patient factors**
 - Ventilatory drive (inspiration)
 - Ventilatory requirements (how much flow and volume required)
 - Timing of the circuits generating the respiratory rhythm (I:E ratio)
- **Ventilator factors**
 - Inspiratory trigger (flow, volume or pressure)
 - Delivery mechanism (flow, volume or pressure)
 - Cycling criteria (when ventilator stops assisting inspiration and allows passive exhalation)

TYPES OF VENTILATOR DYSSYNCHRONY

- **Ineffective triggering**
 - High PEEP (must generate enough effort to overcome PEEP)
 - Weakness
 - Incorrect ventilator settings
 - Ventilator dysfunction
- **Inappropriate triggering** (patient inspires while the ventilator cycles to expiration)
 - Inspiratory time too short
 - Inspiratory flow rate too low
 - Set tidal volume low
 - Coughing and hiccups
- **Autotriggering** (important to distinguish from ineffective triggering)
 - Hiccups, Coughing
 - Cardiac oscillations
 - Shivering, Seizures
 - 'Rain out' (condensation in ventilator circuit)
 - Trigger sensitivity set too low
- **Flow dyssynchrony** (too fast or too slow)
 - **Too slow:** 'pull down' on pressure curve upstroke during inspiration
 - **Too fast:** e.g. discomfort from rise time too short
- **Exhalation dyssynchrony** (too early or too late)

ASSESSMENT

- **Examination**
 - Work of breathing
 - Respiratory pattern
 - Audible sounds (e.g. Cuff leak, stridor, wheeze)
 - Chest findings (e.g. hyperexpansion, dullness, crackles)
- **Monitor**
 - Vital signs
 - ETCO₂
 - SPo₂
- **Ventilator**
 - Waveforms
 - Alarms
- **Chest x-ray**

ED MANAGEMENT OF A PATIENT FIGHTING A VENTILATOR

- **Resuscitation**
 - Address life threats
 - Disconnect patient from ventilator and replace with BVM if required
- **Address patient factors**
 - Treat patient's respiratory pathology affecting resistance and/ or compliance (e.g. Sputum, bronchospasm, chest wall eschar, pneumothorax)
 - Treat other patient factors (e.g. Hunger, pain, weakness, sleep, sedation, nutrition, physiotherapy)
- **Correct problems with the endotracheal tube**
 - Kinking
 - Obstruction (e.g. Secretions blocking)
 - Impingement on carina or between cords
- **Correct problems with the ventilator**
 - Choose **appropriate ventilator**
 - Choose **appropriate mode**
 - Ensure sensitivity is **not too low or high**
 - Choose **appropriate ventilator rate**
 - Set **appropriate flow rate**
 - Check that patient **isn't auto-triggering** (cardiogenic oscillations, high sensitivity, circuit leaks, water condensation in the circuit)
 - **Sedate patient** to reduce agitation
 - **Taking over ventilation** if fatigue is apparent
- **Address ineffective triggering**
 - **Address PEEPI (Intrinsic Positive End-Expiratory Pressure)**: — apply increased externally applied PEEP — decrease tidal volume and respiratory rate — increase expiratory time — bronchodilators
 - **Address weakness**: — nutrition — reduce sedation — physiotherapy
 - **Adjust trigger sensitivity** threshold (may lead to inappropriate triggering)
- **Exhalation dysynchrony**
 - Treat underlying patient factors (e.g. COPD, asthma)
 - Adjust exhalation sensitivity or change to time-control cycling between inspiration and expiration or change to a volume-cycled mode.

CHAPTER 22. RESPIRATORY FUNCTION

I. END-TIDAL CAPNOGRAPHY

- **End-tidal capnography** (end-tidal CO_2 , PETCO_2 , ETCO_2) refers to the graphical *measurement of carbon dioxide partial pressure (mm Hg) during expiration*.
- The American Society of Anesthesiologists (ASA) endorses end-tidal capnography as a standard of care for general anesthesia, moderate sedation, and deep sedation.
- Accordingly, other specialties, including critical care and emergency medicine, are more frequently implementing end-tidal capnography monitoring.

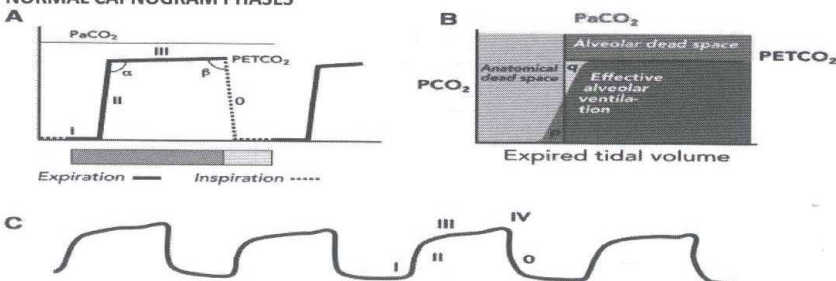
CLINICAL USES OF CAPNOGRAPHY

- *Monitoring Ventilation: Hyperventilation and Hypoventilation.*
- *Confirming, Maintaining, and Assisting Intubation*
- *Measuring Cardiac Output during CPR*
- *Monitoring Sedated Patients*
- *Ventilating Head Injured Patients*
- *Perfusion Warning Sign*

1. CAPNOGRAPHY WAVEFORM INTERPRETATION

- Capnography waveform interpretation can be used for diagnosis and ventilator-trouble shooting.
- The CO_2 waveform can be analyzed for 5 characteristics:
 - *Height/ Frequency/ Rhythm / Baseline / Shape*

NORMAL CAPNOGRAM PHASES



4 PHASES:

- **Phase I (inspiratory baseline):** beginning of exhalation, CO_2 level is ZERO.
- **Phase II (expiratory upstroke):** alveolar gas begins to mix with the deadspace gas and the CO_2 rises rapidly.
- **Phase III (alveolar plateau):** elimination of CO_2 from the alveoli; usually.
- **Phase 0 (inspiratory downstroke):** the beginning of the next inspiration

OTHER FEATURES:

- Normal end-tidal PCO_2 is approximately: 38 mmHg or 5%
- **The alpha angle** is the transition from Phase II to Phase III
- **The beta angle** is the transition from Phase III to Phase I (the start of inspiration)
- An additional phase IV (terminal upstroke before phase 0) may be seen in pregnancy
- ETCO_2 only represents alveolar CO_2 when a relatively horizontal plateau phase (phase III) is seen.

2. CAUSES OF ABNORMAL ETCO2

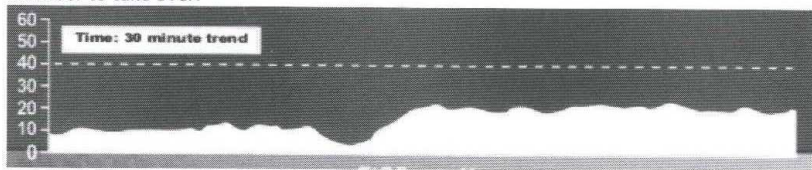
FLAT ETCO2 TRACE	
<ul style="list-style-type: none"> • Ventilator disconnection • Airway misplaced – extubation, oesophageal intubation • Capnograph not connected to circuit • Respiratory/Cardiac arrest • Apnoea test in “brain death” dead patient • Capnography obstruction 	
INCREASED ETCO2	DECREASED ETCO2
CO2 Production	CO2 production
<ul style="list-style-type: none"> • Fever • Sodium bicarbonate • Tourniquet release • Venous CO2 embolism • Overfeeding 	<ul style="list-style-type: none"> • Hypothermia
Pulmonary perfusion	Pulmonary perfusion
<ul style="list-style-type: none"> • Increased cardiac output • Increased blood pressure 	<ul style="list-style-type: none"> • Reduced cardiac output • Hypotension • Hypovolemia • Pulmonary embolism • Cardiac arrest
Alveolar ventilation	Alveolar ventilation
<ul style="list-style-type: none"> • Hypoventilation • Bronchial intubation • Partial airway obstruction • Rebreathing 	<ul style="list-style-type: none"> • Hyperventilation • Apnea • Total airway obstruction • Extubation
Apparatus malfunctioning	Apparatus malfunctioning
<ul style="list-style-type: none"> • Exhausted CO2 absorber • Inadequate fresh gas flows • Leaks in ventilator tubing • Ventilator malfunctioning 	<ul style="list-style-type: none"> • Circuit disconnection (note low airway pressures) • Leaks in sampling tube • Ventilator malfunctioning
SUDDEN DROP IN ETCO2 TO ZERO “DOPES”	SUDDEN INCREASE IN ETCO2
<ul style="list-style-type: none"> • Displacement/ Disconnection • Obstruction/ Pneumothorax • Equipment failure, • Breath Stacking 	<ul style="list-style-type: none"> • ROSC during cardiac arrest • Correction of ET tube obstruction
SUDDEN CHANGE IN BASELINE (NOT TO ZERO)	ELEVATED INSPIRATORY BASELINE
<ul style="list-style-type: none"> • Calibration error • CO2 absorber saturated: check capnograph with room air • Water drops in analyzer or condensation in airway adapter 	<ul style="list-style-type: none"> • CO2 rebreathing (soda lime exhaustion) • Contamination of CO2 monitor (sudden elevation of baseline and top line) • Inspiratory valve malfunction (elevation of the baseline, prolongation of downstroke, prolongation of phase III)

3. HOW TO ANALYZE THE WAVEFORM?

- Use an algorithm or systematic process for analysis. This can be divided into several steps:
 - Look for presence of exhaled CO₂ (Is a waveform present?)
 - Inspiratory baseline (Is there rebreathing?)
 - Expiratory upstroke (What is the shape i.e. steep, sloping, or prolonged?)
 - Expiratory/alveolar plateau (Is it sloping, steep, or prolonged?)
 - Inspiratory downstroke (Is it sloping, steep, or prolonged?)
 - Ensure you evaluate the height, frequency, rhythm, baseline, and shape. With these thoughts in mind, let's discuss some clinical scenarios.

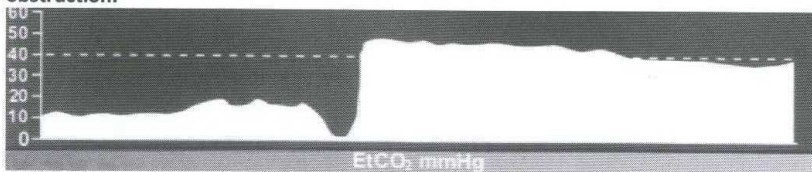
CLINICAL CASES...

- Before you can reassess your other two patients, you receive an EMS radio call. They were called to the scene of a patient in PEA, and they have started compressions and will be at your doorstep in 3 minutes. The patient arrives, with the crew doing high quality CPR. The patient continues with no pulse, leads and ETCO₂ are connected, one amp of epinephrine is given, and US shows a heart rate of 40 bpm. Your waveform capnography shows 10 mm Hg, and the person completing CPR is tiring. As the team leader, you ask another team member to take over.



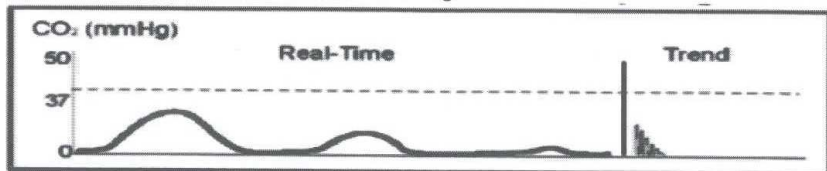
Picture from http://www.slideshare.net/larryide/capnography?next_slideshow=1

- This waveform with a dip shows the time to transition to a different provider, with improved perfusion with the new provider doing compressions, as the CO₂ has increased indicating better tissue perfusion.
- After another minute of CPR, the ETCO₂ jumps to 40.
- A sudden increase in ETCO₂ is seen in ROSC during arrest or correction of an ETT obstruction.

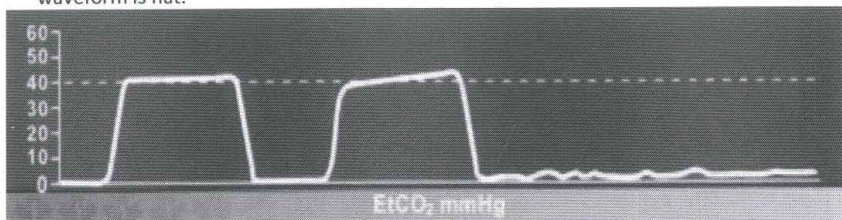


Picture from http://www.slideshare.net/larryide/capnography?next_slideshow=1

- You now have return of pulses and are preparing to intubate the patient.
- Unfortunately, the resident completing it is not confident in his view and is unsure of tube placement. Your waveform shows the following:



- This waveform shows a **tapering of the ETCO₂, suggestive of esophageal intubation.**
- You ask the resident to remove the ETT. He obtains an improved view with videoscope and passes the ETT without difficulty.
- The waveform looks normal, and the patient is now stable.
- Finally, you have time to go reassess your COPD patient. Just as you enter the resuscitation bay, he has a **desaturation to 88% while on FiO₂ of 100%**, and your waveform is flat.

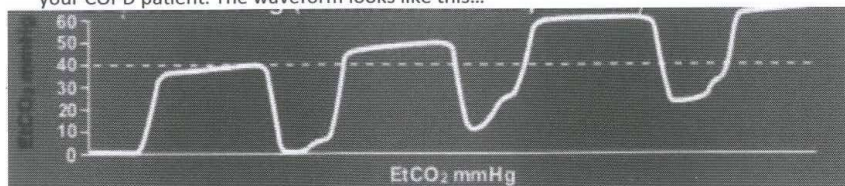


Picture from http://www.slideshare.net/larryide/capnography?next_slideshow=1

- You are now pretty tired of these flat waveforms, and you immediately curb your sphincter response while running to the bedside. Your mind quickly goes through the **DOPES mnemonic** (Displacement/Disconnection, Obstruction, Pneumothorax, Equipment failure, Breath Stacking) and you see that while moving the patient, **the ETT became disconnected from the circuit.** You reconnect, with increase in saturation and good waveform.

WHAT ARE OTHER CAUSES OF A SUDDEN FLAT ETCO₂ TRACING?

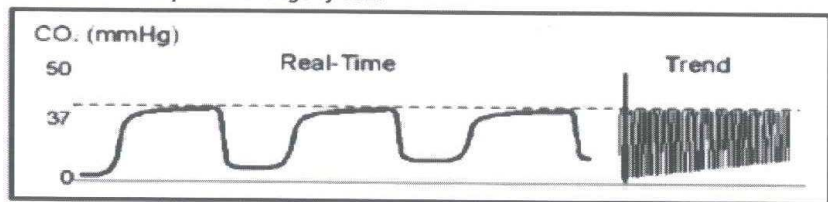
- Extubation,
- Ventilator disconnection
- Capnography not connected to circuit,
- Obstruction of capnography,
- Esophageal intubation.
- Cardiorespiratory arrest,
- Apnea test in brain dead patient,
- After caring for an ankle sprain and beginning the workup of a patient with chest pain, you again reassess the patient with COPD. You notice a steadily increasing EtCO₂ baseline in your COPD patient. The waveform looks like this...



Picture from http://www.slideshare.net/larryide/capnography?next_slideshow=1

- The waveform reflects an **elevation of baseline**, as well as the **plateau**, indicating **incomplete exhalation.** The CO₂ is not being appropriately removed.
- **This is often due to:**
 - Insufficient expiratory time,
 - Inadequate inspiratory flow, or
 - Faulty expiratory valve.

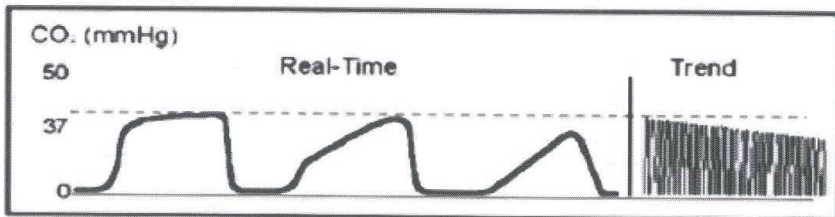
- **Rebreathing** can also appear with the following waveform **with baseline elevation**, which is due **to inadequate exchange of CO₂**.



Picture from http://www.paramedicine.com/pmc/End_Tidal_CO2.html.

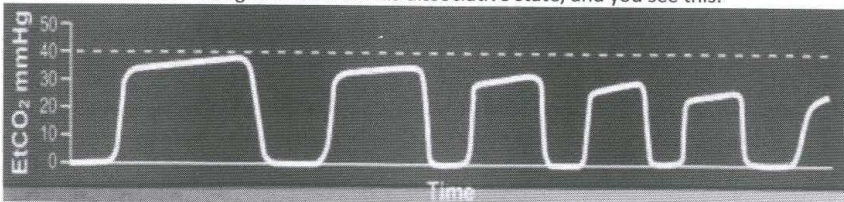
- **INCREASED ETCO₂ CAN BE DUE TO FOUR COMPONENTS:**

- **Increased CO₂ production** (fever, NaHCO₃ administration, tourniquet release, and overfeeding syndrome).
 - **Pulmonary perfusion increase** (increased cardiac output, increased blood pressure).
 - **Alveolar ventilation decrease** (hypoventilation, bronchial intubation, partial airway obstruction, rebreathing).
 - **Equipment malfunction** (exhausted CO₂ absorber, inadequate fresh gas flow, ventilator tubing leak, ventilator malfunction).
- **Once you slow down his respiratory rate and increase the flow rate**, his saturations and **waveform improve**. Suddenly, the alarm alerts you to high pressures in the circuit, and his waveform shows:



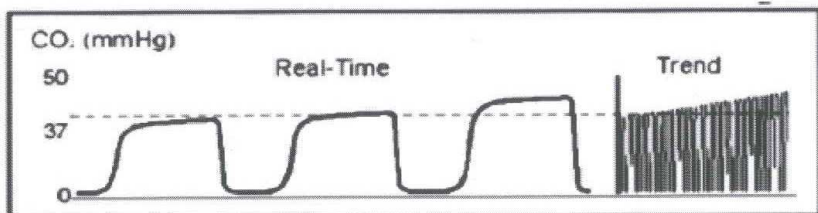
Picture from http://www.paramedicine.com/pmc/End_Tidal_CO2.html

- This waveform is due to **obstruction of the ETT**, either through **ETT kink, foreign body in airway, bronchospasm, or mucous plug**.
- You see high peak pressures and suction the tube, while ordering an in-line duoneb.
- Five minutes later the **patient again improves**.
- After all this excitement, you prepare for **the sedation of the 8-year-old male** with forearm fracture requiring reduction. The sedation and reduction go smoothly with ketamine. He is starting to wake from his dissociative state, and you see this:



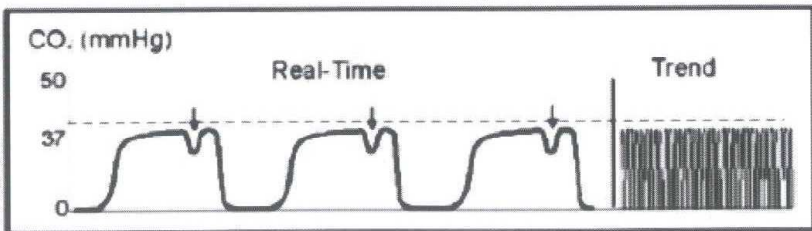
Picture from http://www.slideshare.net/larryide/capnography?next_slideshow=1

- This waveform demonstrates **hyperventilation**.
- **Notice the baseline is unchanged.** This waveform shows steadily decreasing plateau, reflecting **tachypnea**, increase in tidal volume, decreased metabolic rate, or fall in body temperature.
- **A DECREASING ET_{CO}2 HAS SEVERAL ETIOLOGIES:**
 - **Decreased CO₂ production** (hypothermia)
 - **Pulmonary perfusion decrease** (reduced cardiac output, hypotension, pulmonary embolism, cardiac arrest)
 - **Alveolar ventilation increase** (hyperventilation, apnea, total airway obstruction, extubation)
 - **Apparatus malfunction** (circuit disconnection, leak in sampling, ventilator malfunction)
- **WHAT IF HIS RESPIRATORY RATE HAD STARTED TO DECREASE?**
- The alveolar plateau will begin to steadily increase, which is due to decrease in respiratory rate, decreased tidal volume, increased metabolic rate, and hyperthermia.
- Notice the baseline is still close to 0, so CO₂ is appropriately exchanged.



Picture from http://www.paramedicine.com/pmc/End_Tidal_CO2.html

- Just before you send the COPD patient to the ICU, the nurse grabs you, as the waveform has now changed.



Picture from http://www.paramedicine.com/pmc/End_Tidal_CO2.html

- This small dip in the alveolar plateau is known as a **"curare cleft."**
- This waveform appears when the **paralytic begins to subside and the patient tries to breathe during partial paralysis.**
- You increase the analgesic drip, and the patient is transferred to the ICU.

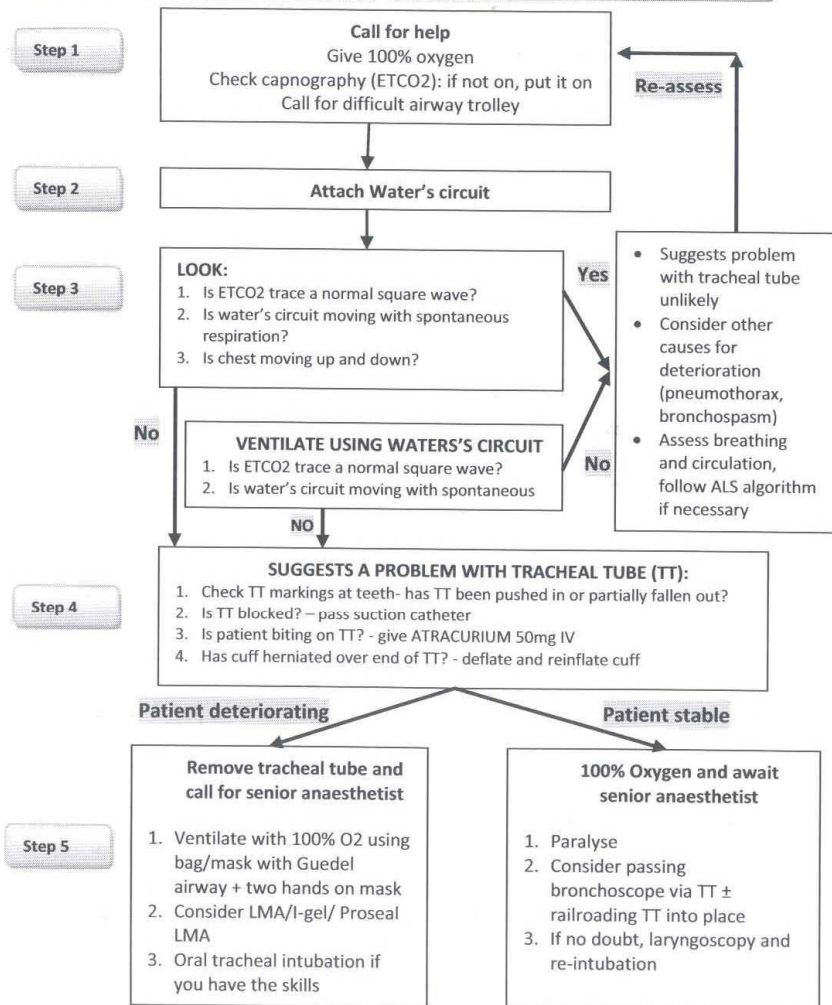
CHAPTER 23. ACCIDENTAL DISPLACEMENT OF TRACHEAL TUBE OR TRACHEOSTOMY

I. TRACHEAL TUBE DISPLACED

You have concern that tracheal tube is displaced:

Consider if:

1. Hypoxia, CVS instability, failure to achieve set pressure/ ventilation
2. Audible cuff leak despite appropriate cuff pressures



II. TRACHEOSTOMY TUBE DISPLACED**You have concern that tracheostomy is displaced:****Consider if:**

1. Hypoxia, CVS instability, failure to achieve set pressure/ ventilation
2. Patient talking despite tracheostomy cuff inflated
3. Audible cuff leak despite appropriate cuff pressures

Step 1

Call for help
 Give 100% oxygen
 Check capnography (ETCO₂): if not on, put it on
 Call for difficult airway trolley

Re-assess**Step 2****Attach Water's circuit****Step 3****LOOK:**

1. Is ETCO₂ trace a normal square wave?
2. Is water's circuit moving with spontaneous respiration?
3. Is chest moving up and down?

Yes**Yes****No****No****Try 2 careful breaths with water's circuit**

1. Is ETCO₂ trace a normal square wave?
2. Is chest moving up and down and easy to ventilate?

No**LOOK AT NECK**

Is it swelling or developing surgical emphysema with each breath?

Yes

- Suggests tracheostomy displacement unlikely
- Consider other causes for deterioration (pneumothorax, bronchospasm)
- Assess breathing and circulation, follow ALS algorithm if necessary

If in doubt

Step 4

SUGGESTS A PROBLEM WITH TRACHEOSTOMY:

1. Is tracheostomy blocked? – pass suction catheter via tracheostomy, ensure inner tube removed
2. Has cuff herniated over end of tracheostomy? – deflate and reinflate cuff

If in doubt

If in doubt or patient deteriorating

If in doubt or patient deteriorating

Fibreoptic inspection via tracheostomy (senior help):

1. Look for tracheal rings and carina
2. Consider advancement over bronchoscope (with great care)

Step 5

DEFLATE TRACHEOSTOMY CUFF AND REMOVE TRACHEOSTOMY

- Cover tracheostomy with sterile gauze and occlusive dressing
1. Ventilate with 100% O₂ using bag and facemask with Guedel airway and two hands on mask
 2. Consider LMA/l-gel/ Proseal LMA
 3. Intubate if you have the skills

When senior help arrives consider:

1. GEB guided insertion of tracheostomy (extreme care if tracheostomy tract <7 days old)
2. RSI and oral reintubation

CHAPTER 24. FLUID CHALLENGE

OVERVIEW

- The assessment of fluid responsiveness is controversial. A fluid challenge remains the gold standard for assessing fluid responsiveness, indeed it is part of the definition...
- **"Fluid responsiveness is an increase of stroke volume of 10-15% after the patient receives 500 ml of crystalloid over 10-15 minutes"** (according to Paul Marik)
- The main drawback of the fluid challenge is the **risk of fluid overload** — particularly in patients undergoing repeated fluid challenges
- Vincent has described the **TROL approach** to safely perform a fluid challenge: Type of fluid (T), Rate of fluid administration (R), Objectives (O) and Limits for safety (L).

1. TYPE OF FLUID

- Dependent on clinical situation (haemorrhage -> use blood, otherwise use a balanced salt solution)
- No differences in mortality in critically ill patients between saline and albumin (SAFE study) -> albumin may be useful in sepsis, avoid in traumatic brain injury
- Avoid HES (starch)
- Avoid excessive chloride administration

2. RATE OF FLUID ADMINISTRATION

- Give 500mL over 10-30 minutes
- No hard data to support a particular regimen
- Recommendations in the Surviving Sepsis Campaign Guidelines:
 - 250-500mL colloid
 - 500-1000mL or 30mL/kg crystalloid

3. OBJECTIVES

- **Options**
 - **Target MAP:** 65mmHg usually OK, may need to higher for the hypertensive patient
 - **Target U/O:** 0.5mL/kg/hr
 - **Resolution of end-organ malperfusion:** resolution of tachycardia, improved LOC, falling lactate, rising ScvO₂ (aim > 70%)
 - **Echo:** optimal filling state (atrial pressures, IVC diameter, ventricular filling and EF)
 - **Cardiac output monitoring** (CO, SV, SVV)
- Some of these are only surrogates for stroke volume and cardiac output, which is what we really want to assess.
 - The utility of surrogates is controversial
 - Some would argue that if tachycardia settles and hypotension resolves during the fluid challenge then you don't need to measure cardiac output.
- **Problems**
 - In practice fluid challenge is limited by the difficulty controlling for other factors that may affect the objective
 - e.g. blood pressure will increase if sedation is changed or the patient is suctioned

4. LIMITS FOR SAFETY

- **Upper limit or increment of CVP or PAWP:** CVP 2-5mmHg increase or PCWP 3-7mmHg
 - if Increase -> stop fluid challenge.
- **Where no invasive monitoring:** measure JVP and look for signs of APO (or use IVC ultrasound and lung ultrasound to assess for 'fluid tolerance')
- If the limit is reached before the objective, the fluid challenge is stopped.



COMMON COMPETENCES

7 QUESTIONS

**Compiled & Edited by:
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ED Registrar**

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CHAPTER 1. HISTORY TAKING

- **AMPLE OR SAMPLE system:**

- **S** - Signs/symptoms
- **A** - Allergies
- **M** - Medications
- **P** - Past medical history
- **L** - Last oral intake/ LMP
- **E** - Events leading to the illness or injury

- **S: Symptoms and signs**

- The time of injury/illness onset,
- What date and how, e.g. "tripped over loose paving stone";
- Where specified, e.g. "on Washington Street";
- Mechanism of injury, e.g. "fell on out-stretched hand and strained the right knee";
- List, numerically, all the injured sites;
- Specify whether a head injury or loss of consciousness (HI/LOC) has been sustained.
- Mention the reason for any delays and how the patient arrived in hospital.
- Note whether there has been a relevant problem before, e.g. "sustained fractured wrist 3 weeks ago; now complaining of (c/o) problem with the plaster (POP)".

- **A: Allergies:**

- "Have you ever had an allergic reaction to anything?"
- "Have you ever had a bad reaction to any drugs?"

- **M: Medication or Treatment:**

- "Do you normally take any medication from your own doctor or from the chemist or do you take the Pill?";
- "Do you take any medicines regularly - pills, patches, inhalers, lotions or creams?"

- **P: Past Medical History:**

- Ask the following leading questions - "have you ever been in hospital before?"
- Have you ever had any operations?
- Have you ever had a serious illness?
- Are you attending an out-patient clinic or your GP presently?
- Have you ever been to an Emergency Department before?"

- **L: Last meal/ Last Hospital visit/LMP:**

- "Do you normally enjoy good health?"
- Have you had any problems recently with your asthma/blood pressure/angina/rheumatism?" (etc).
- "When was your last Tetanus injection?"
- Do you ever remember having such an injection?"
- Last menstrual Period to any childbearing age patient?

- **E: Event/ Social History:**

- "What's your occupation?";
- "Who is at home with you?" (i.e. to do any chores if you are kept in).
- Living alone, at Home or Nursing/Community facility?
- Smoking in pack-year-history
- Illicit drugs use
- Alcohol

CHAPTER 2. CLINICAL EXAMINATION

- **Vital signs:**

- **ECS** (Edinburgh Coma Score): e.g. "ECS 1" in the poisoned patient.
- **RR** (Respiratory Rate): e.g. "RR 28, laboured" in the victim of thoracic trauma.
- **BP** (Blood Pressure): e.g. "BP 190/95 right arm; 130/90 left arm" in chest pain.
- **P** (Pulse): e.g. "P 120 A. Fib."
- **rT** (Rectal Temperature): e.g. "rT32.5c, hypothermic" in the unconscious.
- **SPO₂** (Capillary Oxygen Saturation): e.g. "98% on room air" in pneumothorax case.
- **BM** (Boehringer Mannheim capillary glucose): e.g. "BM 2 (hypoglycaemic)".
- **CNS** (Central Nervous System or Cranial Nerves): e.g. "CNS intact".
- **PERLA** (Pupils equal and reactive to light and accommodation): e.g. in coma case.

- **Then work down meticulously from the head to the extremities**

- **H&N** (Head and Neck): Ideally draw the head and illustrate areas of injury with reasonably precise location and dimensions.
- **CHEST**: Utilise conventional medical diagrams and mention relative positives and negatives, e.g. "lungs clear, normal vesicular breath sounds, normal heart sounds, nil added, no crepitus, etc".
- **ABDOMEN**: Utilise conventional medical diagrams, mention organomegaly, masses, peritonism, bowel sounds, external deformity/swelling/dyscolouration - patterning, genitalia, rectal and vaginal examination, etc.
- **PELVIS**: Mention separately in trauma patients, particularly bleeding.
- **LIMBS**: Specify whether upper or lower, right or left. Mention relevant negatives/ co-incident findings, e.g. "no neurovascular or tendon deficit (no NVTG); no clubbing; palmer erythema; tattooing, injection track-marks, old self-inflicted wounds, etc". Use diagrams as a matter of routine.
- **Neurology**: Specifically mention positive or negative findings: i.e. tone, power, reflexes, sensation, co-ordination or stipulate "no focal neuropathology".
- **Plantars**: Specifically mention the (Babinski) plantar responses, left and right.

- **P (Plan or Procedure):**

- Indicate clearly, in order, list what you intend to do/want done and tick them when they are done, e.g. surgical toilet, sutures (how many), POP (what kind/which limb), medication (specify dose and for how long), brace (e.g. Futura wrist splint) stick/crutches, DTG (double tubigrip), BAS (broad arm sling), C&C (collar and cuff) and investigations (e.g. WTU / FBC / Plasma Chemistry / BM / ABGs /Amylase / CXR / ECG etc).

- **Then sign your name clearly. Record the time you finished.**

- It is imperative that you note the time injured, the time seen by you and the time referred to a colleague. Noting these times has major forensic importance, for the patient and you. Do it and avoid grief!

- The system described here will save you time, sleep and a considerable amount of writing later once it has become a "brain-stem function"

CHAPTER 3. SAFE PRESCRIBING

I. REVERSING WARFARIN

- Bleeding while on oral anticoagulants increases significantly with INR (International Normalised Ratio) level > 5.0, particularly in patients with risk factors for bleeding.
- **RISK FACTORS FOR BLEEDING INCLUDE:**
 - Age (> 70) years
 - Previous bleeding complications
 - Recent initiation of anticoagulants
 - GI haemorrhage/ulcers
 - History of CVA
 - Recent surgery
 - Uncontrolled BP
- The majority of over-anticoagulated patients will return to their target therapeutic range within 3 days of discontinuing warfarin therapy.

1. VITAMIN K

- Vitamin K1 (C₃₁H₄₆O₂) and K2(C₄₁H₅₆O₂) are two naturally occurring fat-soluble vitamins.
- Vitamin K is essential in the production of prothrombin.
- **Vitamin K is the first drug of choice to be administered for the reversal of excessive anticoagulation if the patient has evidence of bleeding.**
- Vitamin K is dispensed in ampoules of 1ml/10mgs known as **Konakion®** or 0.2mls/2mgs known as **paediatric Konakion®**.
- This can be administered sub-lingually using a 1ml syringe and a filter needle to draw up and administer the solution.
- Vitamin K is also available in 10mg tablets for oral administration.
- When partial correction is required to achieve a target therapeutic INR, the Intravenous preparation of Vitamin K can be administered in low doses **of 1-2mgs sub-lingually**.
- **5mgs of Vitamin K will completely reverse anticoagulation**, which is only indicated if the patient is presenting with bleeding as a result of a high INR.
- **Particular caution is advised for patients with prosthetic heart valves**, where the use of vitamin K may increase the risk of thrombosis due to overcorrection of the INR. Therefore, if indicated, **small doses of vitamin K only (e.g. 1 – 2 mg) are recommended**.

2. PROTHROMBIN COMPLEX CONCENTRATE

- It is **not** routinely administered to reverse excessive anticoagulation in the absence of bleeding but **should** be administered **in life threatening major haemorrhage**.
- PCC is more effective than **Fresh Frozen Plasma (FFP)** for reversal of bleeding associated with excessive anticoagulation; **therefore, FFP is not indicated or recommended when PCC is available**.

PROCEDURE

INR	Action
3 < INR < 5	<ul style="list-style-type: none"> • ↓ Warfarin dose or stop (for 1-2 days). • Restart Warfarin (reduced dose) when INR < 5.
5 < INR < 8 No bleeding	<ul style="list-style-type: none"> • Stop Warfarin. • Recheck INR after 1-2 days. • If there are risk factors for bleeding recheck INR level within 24hrs. • Restart warfarin at reduced dose when INR < 5.0 • Determine if there are any causative or contributing factors for the increase in the INR level and adjust dose accordingly.
5 < INR < 8 Minor bleeding	<ul style="list-style-type: none"> • Stop warfarin for 1-2 days. • Consider administration of low dose Vitamin K 1-2mgs sub-lingually, depending on extent of bleeding and risk factors for further bleeding. A low dose of vitamin K e.g. 1-2mgs of the paediatric intravenous preparation, can be administered sub-lingually. For patients with prosthetic heart valves caution should be taken to avoid over correction of the INR below therapeutic range. • Restart warfarin at reduced dose when INR < 5.0 • Determine if there are any causative or contributing factors for the increase in the INR level and adjust dose accordingly • If INR over corrected contact Haematology for dosing instructions and advise.
INR > 8.0	<ul style="list-style-type: none"> • Stop Warfarin • Identify additional risk factors for bleeding: increasing age (e.g. > 70 yr), previous bleeding event/complications (ulcers, wounds, post surgery) • Check for evidence of minor bleeding: epistaxis, bleeding gums, haematuria, oozing wounds, haemoptysis, PR bleeding. • Administer 1-2mgs of Vitamin K sub-lingually. A low dose of vitamin K, e.g. 1-2mgs of the paediatric intravenous preparation, can be administered sub-lingually. For patients with prosthetic heart valves caution should be taken to avoid over correction of the INR below therapeutic range. • Recheck INR within 24hrs and restart warfarin at a reduced dose once INR < 5.0. • If there are no risk factors identified or there is no evidence of minor bleeding recheck INR within 24hrs. • Determine if there are any causative or contributing factors for the increase in the INR level and adjust dose accordingly. • If INR remains > 8.0 after 24hours the dose of Vitamin K can be repeated. • If INR over corrected contact Haematology for dosing instructions and advise.

MAJOR OR LIFE-THREATENING BLEEDING

- *Intracranial bleed.*
- *Retroperitoneal bleed/Intra-ocular bleed.*
- *Muscle bleed, with compartment syndrome.*
- *Pericardial bleed.*
- *Active bleed with hypotension or 3g fall in Hb.*

EMERGENCY REVERSAL

- Stop warfarin
- Consult with Haematology and Cardio-thoracic consultant if mechanical valve in-situ.
- Administer **5mgs of Vitamin K intravenously** (IV Vitamin K will provide 70% correction of INR within 8 hours).
- Administer **Prothrombin Complex Concentrate (PCC) Octaplex as per the manufactures instructions.**

PCC (Octaplex) dose in Major Bleed

Patients INR	Dose PCC
INR 2 - 3.9	25 IU / Kg
INR 4 - 6	35 IU / Kg
INR > 6	50 IU / Kg

- For patients with prosthetic heart valves caution should be taken to avoid over correction of anti-coagulation below therapeutic range. A low dose of **IV Vitamin K (1-2mgs)** can be administered sub-lingually.
- Discuss with cardio-thoracic, cardiac or haematology consultant or registrar before administering Vitamin K.
- Note: there may be an increased risk of bleeding when obtaining intravenous access due to high INR. Recheck **INR within 30-mins to 1 hour** of administration of PCC.
- There may be an initial correction of the INR shortly after administration of PCC however this may be temporary due to the half-life of factor VII in PCC.
- **The INR should be repeated 6hrs post administration of PCC** and regularly until the patients INR is within their target range.
- *Further Vitamin K may be required.*
- *Warfarin should be commenced once haemodynamically stable.*
- If INR over corrected contact Haematology for dosing instructions and advise.

II. REVERSING DABIGATRAN (PRADAXA®)

- Dabigatran is an oral **DIRECT THROMBIN INHIBITOR (DTI)** licensed for stroke prevention in atrial fibrillation. FDA Approves Praxbind® (idarucizumab), Specific Reversal Agent for Pradaxa® (dabigatran etexilate)
- The **PT/INR** response to Dabigatran is inconsistent and should not be measured when assessing a patient who is bleeding or needs emergency surgery.
- The activated partial thromboplastin time (APTT) provides a qualitative measurement of the anticoagulant effect of Dabigatran. Knowledge of the time of last dose is important for interpretation of the APTT.
- **If a patient receiving Dabigatran presents with bleeding:**
 - Omit/delay next dose of Dabigatran
 - Measure APTT and PT (consider DTI assay if available).
 - Administer **activated charcoal, with sorbitol, if within 2 h of ingestion.**
 - Give **Tranexamic acid (TXA)** - 1 g intravenously if significant bleeding
 - Involve haematology team.
 - Maintain renal perfusion and urine output to aid Dabigatran excretion.
 - Dabigatran exhibits low protein binding and may be removed by dialysis.
 - Supportive care should form the mainstay of treatment.
- If life/limb threatening bleeding, consider another haemostatic agent.

III. REVERSING RIVAROXABAN (XARELTO®)

- The PT/INR / APTT responses to Rivaroxaban are not reliable indicators of the level of anticoagulation.
 - Food and Drug Administration (FDA) did not approve the antidote (**AndexXa**) for uncontrolled bleeding linked to blood thinners including **Xarelto (rivaroxaban)** and **Eliquis (apixaban)**.
 - The FDA designated Portola's drug, AndexXa (andexanet alfa), a Breakthrough Therapy and granted it expedited review. But the agency said it needs more information before approval.
 - *Currently (2016), there is no reversal agent for uncontrolled bleeding linked to these drugs.*
 - **Internal bleeding** adverse events linked to Xarelto include fatal brain and stomach bleeds.
- **If a patient receiving Rivaroxaban presents with significant bleeding:**
 - *Omit/delay next dose of Rivaroxaban.*
 - *Administer **activated charcoal**, with sorbitol, if within 2 h of ingestion.*
 - *Give **PCC** at 50 IU / Kg.*
 - *Involve haematology team.*
- **AO DEVICES** potentiates effect of warfarin
- **PC BRAS** decreases effect of warfarin

Interactions of Warfarin



**Liver enzyme (CPY) inducers
(INR reduction)**

PC BRAS

Phenytoin
Carbamazepine
Barbiturates
Rifampacin
Alcohol Excess
Sulphonylurea

**Liver enzyme (CPY) inhibitors
(INR elevation)**

AODEVICES

Amiodarone and Allopurinol
Omeprazole
Disulfiram (metronidazole)
Erythromycin (and clarithromycin)
Valproate
Isoniazid
Cimetidine (and Ciprofloxacin)
Acute Ethanol intoxication
Sulphonamide

DRUGS WITH NARROW THERAPEUTIC RANGE

Renally excreted:

- Digoxin
- Gentamycin
- Vancomycin
- Lithium
- (Metformin): in theory

Hepatically metabolised:

- Phenytoin
- Ciclosporin

CHAPTER 4. PATIENT SAFETY IN CLINICAL PRACTICE

- The ED setting itself undoubtedly contributes significantly to the risk of harm.
- This article illustrates and discusses ED patient-safety issues, and offers some recommendations for improvement in care and prevention of harm.

1. THE ED SETTING

- The ED is unlike any other area of the hospital or health-care setting.
- Patients seek care for both primary care and urgent care complaints at any time of the day or night, on any day of the week, when no other source of care is available.
- The ED setting is fast-paced and requires quick thinking, a broad depth of knowledge about many medical conditions, and a broad range of skills to perform emergent and life-saving procedures.
- Often, patients are presenting to a hospital ED for the first time, with incomplete medical records.
- They may not know their medical conditions or medications, or be in a position to communicate this information.
- Any of these situations alone can lead to an adverse event; in combination, they can significantly **increase the risk for harm**.
- In addition, **ED overcrowding** due to limited availability of inpatient hospital beds may consume resources and staffing needed to care for active ED patients and new patients coming through the door.
- Safety factors in the ED can be categorized as those related to patients, providers, or the environment/systems.

2. SAFETY FACTORS IN ED:

PATIENTS-RELATED	PROVIDER-RELATED	ENVIRONMENT/SYSTEM -RELATED
Acuity and Emergent conditions	Experience	Inadequate staffing
Age extremes	Fatigue	Inexperienced staff
Communication barriers	Shift work	Teamwork and communication problems
Vague or atypical complaints	Cognitive errors	Overcrowding
Undifferentiated presentations	Missed test results	Boarding and admitted patients
Mental status changes	Procedural errors	Lack of equipments
Cognitive impairment	Policy deviation	Equipments failure
Complex medical conditions	Transitions of care	Reliability or inadequate of consultation services
Lack of knowldge of Medical problems and medications		Lack of complete medical records
		Difficulty using electronic health record or order entry system

3. CULTURE OF SAFETY

- Developing and maintaining a “culture of safety” is a commitment to minimize adverse events when performing high-risk jobs that can result in harm.
- This concept originated in other industries such as the airline and nuclear energy industries.

- Organizations and companies are considered high-reliability organizations (HROs) when they are dedicated to preventing harm at all staff levels—from the frontline to the corporate level.
- These HROs promote the reporting of errors and “near misses” without fear of blame or loss of employment.
- In the ED, a culture of safety encourages teamwork, event reporting, communication openness, transparency with feedback and learning from errors, and administrator collaboration for safety.
- In EDs with a strong safety culture, near misses are more likely to be intercepted to reduce patient harm. Teamwork training improves communication and reduces errors.
- One such program, **Team Strategies and Tools to Enhance Performance and Patient Safety** (TeamSTEPPS), was developed by a joint effort of the US Department of Defense and Agency for Healthcare Research and Quality to promote interprofessional communication between all providers in the hospital.
- This program provides many tools, including one to obtain attention in difficult situations and one to escalate concerns to focus on an important safety issue.
- One ED’s experience with TeamSTEPPS led it to identify specific steps to ensure continued success after the initial start.
- To maintain the high level of teamwork and successful communication, this ED recognized a need for continued champions at all staff levels and all new staff members were required to go through the training.
- Another important aspect of a strong safety culture is **creating an environment that promotes reporting of adverse events and near misses**.
- The culture should allow a person involved in an adverse event to feel comfortable reporting such events.
- When administration supports such reporting, the true safety problems in the ED are identified and can be targeted for improvement.

4. TRIAGE

- Different triage models have been introduced in the ED with the aim of prioritizing acute patients and thereby facilitating safer care.
- During triage, the patient’s medical priority is evaluated, and this evaluation is used to sort and prioritize the patient’s condition and need for care.
- International and national studies have described the experiences of various triage models and how patient safety can be improved by triage assessments.
- However, there is insufficient scientific documentation to determine whether the safety, reliability and reproducibility differ between the three most common triage models in Sweden: the Rapid Medical Emergency Triage and Treatment System (RETTS), Adaptive Process Triage (ADAPT) and the Manchester Triage Scale (MTS).
- Using team triage in these models, with a specialist physician in the front line decreased the percentage of patients at the ED leaving without being seen (LWBS).
- Shorter decision pathways between physicians and other team members lead to increased patient safety.

5. PATIENT SAFETY RISKS IN THE ED

- Common problems in the ED that are more likely to lead to patient safety risks or patient injuries are **communication failures** and also communication in connection

with handoff and transfers, overcrowding, and a lack of teamwork, rather than medical mishaps or lack of knowledge.

A. COMMUNICATION FAILURES

- Thus, handoff and transition of patients have become the focus of efforts aimed at reducing errors.
- As much as **50% of errors** in communication occur during handoff, and also the shift change is a critical moment.
- The safety aspects of the transfer of patients from the ED to an admitting physician have not been studied thoroughly.
- The working environment, which demands constant multitasking, is a particular challenge.
- The specific factors critical to patient safety are the patient flow rate, workload, "communication and information technology resources and processes" and assignment of responsibility.
- To improve patient safety, it is important to develop communication skills; e.g., **Identification, Situation, Background, Assessment, Recommendation** (or **ISBAR**), which is a communication tool used in many health care settings.

B. OVERCROWDING

- Overcrowding is one of the leading problems and one of the most common patient safety risks at the ED.
- The definition of overcrowding is often referred to as an extreme number addition of patients in the treatment areas beyond the ED's capacity, which can require medical care to be provided in hallways and similar areas.
- Crowding can also be defined as a situation in which the demand for emergency service exceeds the ability to provide care in a reasonable amount of time.
- Crowding increases the work-load and limits the ability to monitor the patients, who may be physically distant from standard care locations.
- The primary cause of overcrowding is **boarding**, the need to hold patients in the ED after they have been admitted to the hospital because no in-patient beds are available.
- This leads to further problems in the ED, such as longer waiting times, and increased suffering for the patients who must wait.
- **The causes of ED crowding** can be divided into three groups:
 - **Input factors:** reflect sources and aspects of patient inflow.
 - **Throughput:** factors reflect bottlenecks within the ED.
 - **Output factors:** reflect bottlenecks in other parts of the health care system that might affect the ED.
- These factors correspond to a conceptual framework for studying ED crowding.
- **Lack of competence in the triage** may lead to further hospitalizations and more problems in output.
- **High number of patient** in turn generates a high demand for hospital beds, and if there are not enough beds, patient safety may be compromised in other parts of the hospital.

CHAPTER 5. INFECTION CONTROL

• BACKGROUND

- The term **Universal Precautions** has now been replaced with **Standard Precautions**.
- Standard precautions are used in conjunction with **Transmission Based Precautions**.
- Transmission based precautions includes **Contact, Airborne and Droplet**

1. CONTACT PRECAUTIONS

- Used for patients who are suspected or known to be infected or colonised with micro-organisms that can be transmitted by direct contact with the patient, his/her environmental surfaces or patient care items, examples include **M.R.S.A, Clostridium difficile, Gastroenteritis and Scabies**. For these patients, the use of **gloves and aprons** are advised for delivery of direct patient care.

2. DROPLET PRECAUTIONS

- Used for patients who are suspected or known to be infected with respiratory micro-organisms transmitted by large particle droplets. Large particle droplets can be generated by the patient coughing, sneezing, talking or undergoing procedures involving the respiratory tract. These droplets do not remain suspended and fall within a close distance of dispersal (approx. 1 metre from the patient) Examples include **Neisseria Meningitidis, Rubella and Mumps**.

3. AIRBORNE PRECAUTIONS

- Used for patient who are suspected or known to be infected with a micro-organism that has the ability to be disseminated by airborne droplet nuclei.
- These have a small particle residue (5 µm or smaller in size) thus can remain suspended for long periods. Examples include **Mycobacterium tuberculosis (TB), Varicella, smallpox, rubeola (measles)**

4. HAND WASHING/DISINFECTION

What Are Your Hands Carrying?

- Micro-organisms found on the skin include two categories:
 - **Resident Micro-Organisms (normal flora)**
 - These are usually deep seated in the epidermis, are not readily removed and do not readily cause infections. However, during surgery/invasive procedures, they may enter deep tissues and establish an infection.
 - **Transient Micro-Organisms**
 - These are organisms that are not part of the normal flora and represent recent contamination, that usually survives for a limited period of time.
 - They are easily removed by a good hand washing technique. They include most of the organisms responsible for cross infection, e.g. Gram-negative bacilli (E.coli, Klebsiella, Pseudomonas spp, Salmonella spp.), Staph aureus, MRSA and viruses e.g. rotaviruses (Damani, N.N. (1997)).

DIFFERENT LEVELS OF HAND HYGIENE

- There are three recommended levels of Hand Hygiene to ensure that the hand hygiene performed is suitable for the task being undertaken. The efficacy of hand hygiene will depend on application of an adequate volume of a suitable hand hygiene agent with good technique for the correct duration of time, and finally ensuring that hands are dried properly.

A. SOCIAL HAND HYGIENE- ROUTINE HAND WASHING

- The aim of social (routine) hand washing with soap and warm water is to remove dirt and organic material, dead skin and most transient organisms. On visibly clean hands it can be undertaken using an alcohol hand rub, and this will remove transient organisms.

B. ANTISEPTIC HAND HYGIENE

- Antiseptic hand disinfection with an antiseptic hand wash agent i.e. Hydrex is generally carried out for aseptic procedures on the ward and for areas of Isolation. Hygienic hand disinfection will remove and kill most transient micro- organisms.
- **Indications for use:**
 - During outbreaks of infection where contact with blood/body fluids or situations where microbial contamination is likely to occur.
 - In "high" risk areas e.g. isolation, ICU etc.
 - Before/after performing an invasive procedure
 - Before/after wound care, urethral or IV catheters etc.

C. SURGICAL HAND HYGIENE

- Surgical hand washing requires the removal and killing of transient micro-organisms and substantial reduction and suppression of the resident flora of the surgical team for the duration of the operation, in case a surgical glove is punctured/torn.
- Ensure that fingernails are kept short and clean.
- Wrist watches and jewellery **MUST** be removed before surgical hand.

5. INFECTIONS CONTROL MEASURES

A. DIARRHOEA

Any patient experiencing diarrhoea should:

- Be **isolated** in a single room with **contact precautions**
- Have a stool record/chart
- Have a stool sample taken for C&S and clostridium difficile culture

B. MRSA

- Patient "**isolated**" in a single room with **contact precautions**
- White coats **OFF** and apron **ON** before entering room
- 7 Day of topical treatment (protocol)
- 3 screens post-protocol (Nose/perineum/axillae/wounds/" drips and drains" devices)
- The patient is only deemed **MRSA negative** when they have had 3 consecutive negative MRSA screens post treatment
- For X-Ray, theatre and Other Department Procedures, please ensure the department is informed of the MRSA status

C. SHARPS INJURY

- Bleed the site, Wash the site under running water
- Complete an incident form
- Attend the Emergency Department (**needlestick policy**) with completed form

D. INTRAVENOUS DRUGS

- Must be re-constituted on the IV tray and administered using aseptic technique
- Gloves must be worn throughout the procedure
- Disposable trays must be used to carry the equipment
- Sharps must **NOT** be carried by hand

6. CLOSTRIDIUM DIFFICILE

- Refer to Major Presentations, Chapter 14

7. METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA)

- Glove Wearing and Hand-washing.
- Notifiable diseases.

RISK FACTORS FOR MRSA COLONIZATION

- | | |
|--|---|
| ○ Recent hospitalization | ○ Men who have sex with men |
| ○ Prolonged hospital stay | ○ Injection drug use |
| ○ Residence in a long-term care facility | ○ Sharing needles, razors, or other sharp objects |
| ○ Recent antibiotic therapy | ○ Sharing sports equipment |
| ○ Hemodialysis | ○ Incarceration |
| ○ HIV infection | ○ Military service |
| ○ Diabetes | |
| ○ Swine farming | |

PARENTERAL ANTIMICROBIAL TREATMENT OF MRSA SKIN AND SOFT TISSUE INFECTIONS

DRUG	ADULT DOSE
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Antibiotics of choice*

Vancomycin	15 to 20 mg/kg/dose every 8 to 12 hours, not to exceed 2 g per dose
Daptomycin	4 mg/kg IV once daily

ALTERNATIVE AGENTS**Short-acting alternative agents with parenteral or oral dosing**

Linezolid	600 mg IV (or orally) twice daily
Tedizolid	200 mg IV (or orally) once daily

Short-acting alternative agents with parenteral dosing

Ceftaroline	600 mg IV every 12 hours
Telavancin	10 mg/kg once daily

Long-acting alternative agents with parenteral dosing

Dalbavancin	Single-dose regimen: 1500 mg once Two-dose regimen: Initial dose 1000 mg, followed by 500 mg dose one week later
Oritavancin	1200 mg IV as a single dose

CHAPTER 6. LONG TERM CONDITIONS CARE

1. HEALTH VISITOR

- The Health Visiting Team **work with all families before or after the birth of a child.**
- Their role is to:
 - Assess the health needs of families, offering support as required.
 - Encourage and support families to make healthy lifestyle choices through health education and health promotion information.
 - Provide information about the many services available to support families.

2. DISTRICT NURSES

- The District Nursing Team will provide high quality, safe and **effective nursing care to Adults who require care delivery in their own home.**

3. MIDWIFE

- Midwives are specialists in normal pregnancy and birth, and their role is to look after a pregnant woman and her baby throughout a phase of antenatal care, during labour and birth, and for up to 28 days after the baby has been born.

4. OCCUPATIONAL THERAPY

- Occupational Therapy provides practical support to people with physical and mental health disability, long term condition, or those experiencing the effects of ageing, to do the things they need or want to do.
- It enables people of all ages to carry out practical and purposeful activities.
- **This could be essential day to day tasks**-such as dressing, cooking, going shopping, to the things that make us who we are – our jobs, interests, hobbies and relationships.
- Occupational Therapy considers all our needs, for example physical, psychological, social and environmental, and helps to increase people's independence and satisfaction in all aspects of life.

5. PHYSIOTHERAPY

- The Physiotherapy service aims to provide the interventions needed to support people **to manage their Musculoskeletal conditions.** This will encompass assessment and treatment, management and advice of wider health issues, managing and supporting people to return to their work or meaningful activities.

6. COMMUNITY MENTAL HEALTH TEAM

- The service provides community focused **psychiatric assessment and treatment interventions for patients from 18 to 65 years of age.** Patients will have access to Nursing Staff, Occupational Therapists, Psychiatrists, Psychologists, Social Work, Area Crisis Service, Primary Care Mental Health Team and the voluntary sector.

7. COMMUNITY REHABILITATION TEAM

- The service provides multi-disciplinary **rehabilitation at home for adults with disabilities and older people.** The service provides Physiotherapy, Occupational Therapy, Nursing Services, Dietetic and Support Services to any adult (over 16years old) with a disability and older people in their own homes. Individuals will go through an assessment process to determine what type of rehabilitation package is necessary for their situation.

CHAPTER 7. PUBLIC HEALTH

I. SCREENING PROGRAMMES

1. ABDOMINAL AORTIC ANEURYSM SCREENING PROGRAMME

- The NHS abdominal aortic aneurysm (AAA) screening programme is available for **all men aged 65 and over** in England.
- The programme aims to reduce AAA related mortality among men aged 65 to 74.
- **A simple ultrasound test** is performed to detect AAA. The scan itself is quick, painless and non-invasive and the results are provided straight away.
- A result letter is also sent to all patients' GPs.

2. BOWEL CANCER SCREENING PROGRAMME

- People eligible for screening receive an invitation letter explaining the programme, along with an information leaflet explaining **the benefits and risks of bowel cancer screening**.
- About a week later, the programme should send a **faecal occult blood sampling kit**. The kit includes simple instructions for:
 - Completing sampling at home. Sending the samples to the laboratory
- The sample is then processed and the results sent to the individual within 2 weeks.
- **Colonoscopy**
 - Healthcare professionals should offer patients with an abnormal screening result a colonoscopy.
 - The **quality assurance guidelines for colonoscopy** explain how to complete the procedure. If the procedure finds polyps, a wire loop passed down the colonoscope tube can remove them and these tissue samples must be tested for any abnormal cells that may be cancerous.
- **Bowel scope screening**
 - The programme is also rolling out **bowel scope screening** to all men and women in **England aged 55**. Healthcare professionals should explain to people that this new test is not yet available everywhere across the country.
 - The target is for all screening centres to be operational by December 2016.

3. BREAST SCREENING

- Eligible women, **aged 50 to 70**, receive an invitation letter explaining:
 - The programme
 - The benefits and risks of breast screening
- Women do not always receive an invitation when they turn 50. They can expect their invitation within 3 years of their 50th birthday.
- Women **cannot walk in and request breast screening unless they are over 70**, when they can request screening every 3 years.
- **Age extension**
 - In some areas, women **aged 47 to 49 and 71 to 73** receive invitations for screening.
 - This is part of a study looking at whether to **extend the breast screening age range**.
- **Higher risk women**
 - Women identified as having a **higher risk of breast cancer** should receive:
 - A formal assessment
 - The opportunity to discuss risk management options

4. CERVICAL SCREENING

- NHS cervical screening programme is available to **women aged 25 to 64** in England.
- All eligible women who are registered with a GP automatically receive an invitation by mail.
- Women **aged 25 to 49** receive invitations **every 3 years**.
- Women **aged 50 to 64** receive invitations **every 5 years**.
- NHS Choices provides information for the public on the **cervical screening programme**.

SCREENING Tests

- Cervical screening is a method of preventing cancer by detecting and treating abnormalities of the cervix.
- **Cervical cytology**
 - The programme uses liquid based cytology (LBC) to collect samples of cells from the cervix.
 - The laboratory will examine these samples under the microscope to look for any abnormal changes in the cells.
- **Human papillomavirus**
 - Human papillomavirus (HPV) is a common virus transmitted through sexual contact. In most cases, a woman's immune system will clear the infection without the need for treatment.
 - HPV has over 100 subtypes, most of which do not cause significant disease in humans.
 - Known as high risk HPV (HR-HPV), some subtypes can cause cervical cancer. In particular **HPV16 and HPV18**.

5. DIABETIC EYE SCREENING

- The eligible population for DES is all people **with type 1 and type 2 diabetes aged 12 or over**.
- People already under the care of an ophthalmology specialist for the condition are not invited for screening.
- The programme offers pregnant women with type 1 or type 2 diabetes additional tests because of the risk of developing retinopathy.
- Screening gives people with diabetes and their primary diabetes care providers **information** about very early changes in their eyes.
- Early warnings allow people to take preventative action to stop serious retinopathy developing.

II. PRINCIPLE OF NOTIFICATION

- Registered medical practitioners (RMPs) have a statutory duty to notify the 'proper officer' at their **local council** or **local health protection team** (HPT) of suspected cases of certain infectious diseases.
- Complete a **notification form** immediately on diagnosis of a suspected notifiable disease.
- **Don't wait for laboratory confirmation** of a suspected infection or contamination before notification.
- Send the form to the proper officer **within 3 days**, or notify them verbally **within 24 hours** if the case is urgent, securely:
 - *By phone*
 - *Letter*
 - *Encrypted email*
 - *Secure fax machine*
- All proper officers must pass the entire notification to Public Health England (PHE) within 3 days of a case being notified, or within 24 hours for urgent cases.

LIST OF NOTIFIABLE DISEASES

- Diseases notifiable to local authority proper officers under the Health Protection **(Notification) Regulations 2010**:
 - Acute encephalitis
 - Acute infectious hepatitis
 - Acute meningitis
 - Acute poliomyelitis
 - Anthrax, Botulism
 - Brucellosis, Cholera, Diphtheria
 - Enteric fever (typhoid or paratyphoid fever)
 - Food poisoning
 - Haemolytic uraemic syndrome (HUS)
 - Infectious bloody diarrhoea
 - Invasive group A streptococcal disease
 - Legionnaires' disease, Leprosy
 - Malaria, Measles, Meningococcal septicaemia, Mumps
 - Plague
 - Rabies, Rubella
 - Severe Acute Respiratory Syndrome (SARS)
 - Scarlet fever, Smallpox
 - Tetanus
 - Tuberculosis
 - Typhus
 - Viral haemorrhagic fever (VHF)
 - Whooping cough
 - Yellow fever

Report other diseases that may present significant risk to human health under the category 'other significant disease'.

CHAPTER 8. ETHICS AND CONFIDENTIALITY

I. CONFIDENTIALITY & SHARING INFORMATION

THE PRINCIPLES OF CONFIDENTIALITY

- Confidentiality is central to the trust between doctors and patients and an essential part of good care. Without assurances about confidentiality, children and young people, as well as adults, may be reluctant to get medical attention or to give doctors the information they need to provide good care.
- Teenagers may be particularly concerned about keeping confidential information from their parents, schools, children's services, the police and other statutory agencies.
- Young people, parents and other adults receiving psychiatric care, and other vulnerable people might have similarly increased concerns about sharing confidential information.
- But sharing information appropriately is essential to providing safe, effective care, both for the individual and for the wider community. It is also at the heart of effective child protection. It is vital that all doctors have the confidence to act on their concerns about the possible abuse or neglect of a child or young person.
- Confidentiality is not an absolute duty. You can share confidential information about a person if any of the following apply:
 - *You must do so **by law or in response to a court order.***
 - The person the information relates to has *given you their consent to share the information* (or a person with parental responsibility has given consent if the information is about a child who does not have the capacity to give consent).
 - It is justified in the **public interest** – for example, if the benefits to a child or young person that will arise from sharing the information outweigh both the public and the individual's interest in keeping the information confidential.

KEY POINTS

- **Tell an appropriate agency** promptly if you are concerned that a child or young person is at risk of, or is suffering, abuse or neglect.
- **Get advice** if you are concerned about the possibility of abuse or neglect, but do not believe that the child or young person is at risk of significant harm.
- **Ask for consent** to share information unless there is a compelling reason for not doing so.
- Information can be shared without consent if **it is justified in the public interest or required by law.**
- Do not delay disclosing information to obtain consent if that might put children or young people at risk of significant harm.
- Tell your patient what information has been shared, with whom and why, unless doing this would put the child, young person or anyone else at increased risk.
- Get advice if you are not sure what information to share, who to share it with or how best to manage any risk associated with sharing information.
- In England and Wales doctors are under a legal duty to report known cases of female genital mutilation in girls and young women aged under 18 to the police.

1. DISCLOSURES WITH CONSENT

- Before disclosing any information to a third party *the patient's consent should be sought.*
- **Consent may be implied**, for example most patient understand that information about their health needs to be shared within the treating healthcare team.

- Implied consent is also acceptable for the purposes of clinical audit, provided patients have been made aware of this possibility by notices in the hospital and have not actively objected. **Express consent** is required if patient-identifiable information is to be disclosed for any other purpose, unless required by law or in the public interest.
- For the consent to disclose information to be valid, patients must be **competent to give consent and provided with information about the extent of the disclosure**.
- If a patient lacks capacity, demonstrated by the functional test of capacity advised in the Mental Capacity Act 2005, then disclosure of information **should be in the patient's best interest**.
- **If a patient, under the age of 16 years**, is able to understand the purpose and consequences of the disclosure (Gillick competent) they can give or withhold consent.
- **If the young person refuses disclosure** but this is necessary to protect the young person from serious harm (e.g. neglect or abuse) this is justifiable. The young person should be made aware of the disclosure and the reasons behind the disclosure.
- **If a young person is not competent to give consent**, someone with parental responsibility may consent to the disclosure on their behalf.
- **In a patient aged 16–17 who lacks capacity**, both the Mental Capacity Act 2005 and the Children Act 1989 can apply, depending on the circumstances.

2. DISCLOSING INFORMATION WITHOUT CONSENT

- If it is probable that a crime has been committed, the police will ask for more information.
- If the patient cannot give consent because, for example, they are unconscious, or refuses to disclose information or to allow you or your colleagues to do so, you can still disclose information if it is required by law or if you believe it is justified in the public interest.
- **Disclosures in the public interest may be justified when:**
 - *Failure to disclose information may put the patient, or someone else, at risk of death or serious harm, or*
 - *Disclosure is likely to help in the prevention, detection or prosecution of a serious crime.*
- If there is any doubt about whether disclosure without consent is justified, the decision should be made by, or with the agreement of, the consultant in charge, or the trust's Guardian.
- If practicable, you should seek the patient's consent to the disclosure, or tell them that a disclosure has been made unless, for example, that:
 - *May put you or others at risk of serious harm, or*
 - *Would be likely to undermine the purpose of the disclosure, by prejudicing the prevention, detection or prosecution of a crime.*
- You must document in the patient's record your reasons for disclosing information without consent and any steps you have taken to seek their consent, to inform them about the disclosure, or your reasons for not doing so.
- If there is no immediate public interest reason for disclosing personal information, no further information should be given to the police.
- The police may seek an order from a judge or a warrant for the disclosure of confidential documents. You should tell those responsible for the continuing care of the patient that further discussion with the patient is needed to ensure, for example, that they are fit to hold a firearms licence.

3. DISCLOSURES REQUIRED IN THE PUBLIC INTEREST

- It should be presumed that **clinical information should not normally be disclosed without the explicit, written consent of the patient.**
- Only information that is directly relevant to the case should be disclosed.
- In certain scenarios, releasing information to the police is in the public interest.
- The decision to release information should be made **by the Consultant in charge**, or his **deputy**. The Consultant in Charge should consider discussing this with another experienced colleague or the **Trust's Caldicott guardian**.
- Disclosure should be considered **where a serious crime has been committed**. 'Serious crime' has not been defined in law, but normally includes; *rape, abuse of a child or vulnerable adult, terrorism, murder and injuries from guns and knives*.
- Theft, burglary, fraud and damage to property are not generally regarded as serious crimes.
- In all cases the balance of breaching a patient's confidentiality and the possible harm caused by this should be weighed against the benefits of disclosing the information.
- The information should be anonymised if possible and the minimum, relevant information only should be disclosed.
- Patient consent should be sought if possible and the patient kept informed of any disclosures, unless this undermines the purpose of the disclosure.
- The ultimate decision about whether or not a disclosure was made in the public interest is determined by the courts. All decisions must be justified and clearly documented.
- A competent adult's wishes should generally be respected if they refuse to allow disclosure and no-one else will suffer. However, if the disclosure is to protect an incompetent patient from serious harm, there is an expectation that the relevant confidential information will be disclosed.
- If such information is not disclosed this will need to be justified.

4. REPORTING GUNSHOT AND KNIFE WOUNDS

- Disclosure of personal information about a patient without consent may be justified in the public interest **if failure to disclose may expose others to a risk of death or serious harm.**
- You should still seek the patient's consent to disclosure if practicable and consider any reasons given for refusal. Such a situation might arise, for example, when a disclosure would be likely to assist in the prevention, detection or prosecution of serious crime, especially crimes against the person.
- **When victims of violence refuse police assistance**, disclosure may still be justified if others remain at risk, for example, from someone who is prepared to use weapons, or from domestic violence when children or others may be at risk.
- *If a patient's refusal to consent to disclosure leaves others exposed to a risk so serious that it outweighs the patient's and the public interest in maintaining confidentiality, or if it is not practicable or safe to seek the patient's consent, you should disclose information promptly to an appropriate person or authority.*
- **You should inform** the patient before disclosing the information, if practicable and safe, even if you intend to disclose without their consent.
- The guidance in *Confidentiality* applies to all violent crime, but gunshot and knife wounds raise issues that warrant special consideration.
- That is not to suggest that information should not be disclosed to assist in the prevention, detection or prosecution of other serious crime.

A. GUIDANCE:

- **This guidance describes a two-stage process:**
 - **You should inform the police quickly** whenever a person arrives with a gunshot wound or an injury from an attack with a knife, blade or other sharp instrument. This will enable the police to make an assessment of risk to the patient and others, and to gather statistical information about gun and knife crime in the area.
 - **You should make a professional judgement** about whether disclosure of personal information about a patient, including their identity, is justified in the public interest.
- The police are responsible for assessing the risk posed by a member of the public who is armed with, and has used, a gun or knife in a violent attack. They need to consider:
 - *The risk of a further attack on the patient*
 - *The risk to staff, patients and visitors in the ED or hospital, and*
 - *The risk of another attack near to, or at, the site of the original incident.*
- For this reason, the police should be informed whenever a person arrives at hospital with a gunshot wound. Even accidental shootings involving lawfully held guns raise serious issues for the police about, for example, **gun licensing**.
- The police should also be informed when a person arrives at a hospital with a wound from an attack with a knife, blade or other sharp instrument. The police should not usually be informed if a knife or blade injury is accidental, or a result of self-harm. If you are in doubt about the cause of the injury, you should if possible consult an experienced colleague.
- *Quick reporting at this stage may help prevent further incidents or harm to others. If you have responsibility for the patient, you should make sure that the police are contacted, but you can delegate this task to another member of staff.*
- Personal information, such as the patient's name and address, should not usually be disclosed in the initial contact with the police. The police will respond even if the patient's identity is not disclosed. The police need to be informed quickly in order to respond to the risk to patients, staff and the public. They also need statistical information about the number of gunshot and knife injuries, and when and where they occur, to inform their own and their crime reduction partners' operational and strategic priorities.

B. MAKING THE CARE OF THE PATIENT YOUR FIRST CONCERN

- When the police arrive, **you should not allow them access to the patient** if this will delay or hamper treatment or compromise the patient's recovery.
- If the patient's treatment and condition allow them to speak to the police, you or another member of the healthcare team should **ask the patient whether they are willing to do so**.
- If they are not, you should explain what the consequences, if any, might be. You, the rest of the healthcare team, and the police must abide by the patient's decision.

C. CHILDREN AND YOUNG PEOPLE

- Any child or young person under 18 arriving with a gunshot wound or a wound from an attack with a knife, blade or other sharp instrument **will raise obvious child protection concerns**. You must inform an appropriate person or authority promptly of any such incident. Knife or blade injuries from domestic or occupational accidents might also raise serious concerns about **the safety of children and young people**.
- You should consider the advice on child protection in **0-18 years: guidance for all doctors** whenever you are concerned that a child may be the victim of abuse or neglect.
- You must be able to justify a decision not to share a concern that children or young people are at risk of abuse, neglect or other serious harm, having taken advice from a named or

designated doctor for child protection or an experienced colleague, or a defence or professional body.

5. PROVIDING A WITNESS STATEMENT FOR THE POLICE

• SCOPE

- This document guides clinicians (Doctors and Emergency Nurse Practitioners) in how to prepare witness statement to use as evidence.
- This document should standardise the content of a witness statement and defines emergency, urgent and standard statements.

• THE STATEMENT

- Clinicians working in emergency departments have an important societal role in assisting the police. A witness statement should be provided promptly after a request by the police. A witness statement is usually related to a patient attending the Emergency Department with injuries due to an alleged assault.
- The statement should only be issued after the patient has provided written consent or a request is issued by a judicial authority.
- The main purpose of a statement is to provide an evidence of facts that will be used in court. The statement is a way of providing evidence in court that is as valid as if the evidence was presented in person. It is for this reason that a declaration attesting to the truth of the statement is signed. Any dishonesty in a signed statement amounts to perjury and may lead to prosecution.
- The statement is a method of communicating medical information to a lay person and so medical terms should be explained in a way that is easy to understand with medical terms explained. It must be noted that an omission could be as improper as an invalid piece of information that is included.
- The police can request personal (not clinical) details regarding attendances to the Emergency Department **if the request is made in writing on Form 826C** and relates to a serious, arrestable crime (Police and Evidence Act 1984) or the Road Traffic Act 1988.
- The form must be signed by an Inspector or above.

A. EMERGENCY STATEMENTS

- There are uncommon occasions when the police request an emergency statement.
- A witness statement concerning a serious crime of violence, injury or death is required by a police officer at the first available opportunity.
- This statement will directly affect the ability of the police to investigate a serious offence or decision to be able to arrest, detain or charge a suspect within the limited time available under the law where appropriate.
- These statements will normally be confined to a description of the injury/injuries and a brief account of the nature of the treatment. ***They should be obtained from the most senior doctor involved in the patient's initial care*** and will be handed to the police without delay. In these circumstances the police will normally make the request due to either the serious nature of the case or because of time and legal constraints relating to a person in custody or whose detention is imminent.
- Where such a request is made, it will be on **the authority of an officer not below the rank of Inspector**, whose name will be provided to the Emergency Department being requested to provide the statement.
- These statements should be regarded as provisional and returned to the police as promptly as possible, **within 12 hours of a request**.

B. URGENT STATEMENTS

- This is a witness statement required to meet a deadline required for a prosecution, breach of which could seriously prejudice the continuation of the proceedings which will usually contain information which details a key element of an offence being charged or prosecuted. This should be provided **within 72 hours of a request**.
- Where the request for an urgent statement is made by the police it will be made on the authority of **The Criminal Justice case manager or the officer in charge of the case**.
- Where the request is made later in proceedings by The Crown Prosecution Service, it will be made by a named lawyer who has responsibility for and is actively reviewing the case.

C. STANDARD STATEMENTS

- These are all other cases where a witness statement is required from hospital medical staff, production of which will normally be not later than **two weeks from the receipt** of the request by the hospital liaison officer.
- The request for such statements will be made by the police via **The Criminal Justice Case Manager or an identified Police Liaison Officer**.

• CONSTRUCTING THE STATEMENT

- The contents of the statement are based on the patient's records and other documents related to his or her attendance.
- Medical history and history of other conditions or illnesses should not be a routine part of the statement unless relevant to the episode of attendance.
- The statement is better typed and a copy stored in a secure computer.
- Hand written statements should be clear, legible, and in black ink.
- A copy should be kept for future reference.

• GIVING OPINION

- A witness statement is a professional statement of facts only. Opinion is given by experts only and this should be based on extensive experience, knowledge, and research. Opinion should be justified and substantiated.

6. DOCTORS GIVING EVIDENCE IN COURT / WITNESS CARE

- The Crown Prosecution Service will make every reasonable effort **to avoid calling a member of the hospital medical staff as a witness** to give oral testimony at the court.
- This will be done, wherever possible, by serving the evidence on the defence and seeking to agree it, or by identifying any issues in contention for further consideration.
- The service of the original medical notes exhibited to a statement, where this can be agreed and arranged will often avoid having to call a member of the medical staff as a witness. Where the original medical records or copies thereof are appended to statements the patient's address and telephone number and any information related to third parties (e.g. identity and addresses of next of kin, relatives or employers) should be removed or suitably obscured in any copy served on or shown to the defence.
- Prompt responses from medical staff for further information when required may also assist in avoiding the calling of such staff to give evidence.
- **When called to testify:**
 - The first duty of all witnesses is to the court.
 - Give evidence that is impartial, honest and not misleading.

- Only give testimony and express opinions about issues that are within your professional competence.
- Work within the timescales set by the court.

7. REPORTING CONCERNS TO THE DVLA

- Confidential medical care is recognised in law as being in the public interest. However, there can also be a public interest in disclosing information: **to protect individuals or society from risks of serious harm**, such as serious communicable diseases or serious crime; or **to enable medical research, education** or other secondary uses of information that will benefit society over time.
- Personal information may, therefore, be disclosed in the public interest, without patients' consent, and in exceptional cases where patients have withheld consent, if the benefits to an individual or to society of **the disclosure outweigh both the public and the patient's interest in keeping the information confidential**. You must weigh the harms that are likely to arise from non-disclosure of information against the possible harm, both to the patient and to the overall trust between doctors and patients, arising from the release of that information.
- Disclosure of personal information about a patient without consent may be justified in the public interest if failure to disclose may expose others to a risk of death or serious harm.
- You should still seek the patient's consent to disclosure if practicable and consider any reasons given for refusal.
- The **Driver and Vehicle Licensing Agency (DVLA)** and **Driver and Vehicle Agency (DVA)** are legally responsible for deciding if a person is medically unfit to drive. This means they need to know if a driving licence holder has a condition or is undergoing treatment that may now, or in the future, affect their safety as a driver.
- You should seek the advice of an experienced colleague or the DVLA or DVA's medical adviser if you are not sure whether a patient may be unfit to drive. You should keep under review any decision that they are fit, particularly if the patient's condition or treatments change.
- The DVLA's publication *Assessing fitness to drive – a guide for medical professionals* includes information about a variety of disorders and conditions that can impair a patient's fitness to drive (See Major Presentations, Chapter 9 / TLoC).
- **The driver is legally responsible for informing the DVLA or DVA** about such a condition or treatment. However, if a patient has such a condition, you should explain to the patient:
 - *That the condition may affect their ability to drive (if the patient is incapable of understanding this advice, for example, because of dementia, you should inform the DVLA or DVA immediately), and*
 - *That they have a legal duty to inform the DVLA or DVA about the condition.*
- If a patient refuses to accept the diagnosis, or the effect of the condition on their ability to drive, you can suggest that they seek a second opinion, and help arrange for them to do so. You should advise the patient not to drive in the meantime.
- If a patient continues to drive when they may not be fit to do so, **you should make every reasonable effort to persuade them to stop**. As long as the patient agrees, you may discuss your concerns with their relatives, friends or carers.
- If you do not manage to persuade the patient to stop driving, or you discover that they are continuing to drive against your advice, **you should contact the DVLA or DVA immediately and disclose any relevant medical information**, in confidence, to the medical adviser.

- Before contacting the DVLA or DVA you should try to inform the patient of your decision to disclose personal information.
- You should then also inform the patient in writing once you have done so.

8. DISCLOSURES AFTER A PATIENT'S DEATH

- The duty of confidentiality to a patient remains after their death.
- There are certain circumstances where disclosure may be justified. For example, responding to a complaint, including those made by bereaved relatives.
- The Access to Health Records Act 1990 allows relevant information to be disclosed to the 'personal representative' of the deceased (usually the executor of the will, or an administrator if there is no will) or anyone who may have a claim arising from the patient's death (e.g. a life insurance claim).
- If the patient requested that specific information remained confidential their views should be respected, subject to those disclosures required by law or justified in the public interest.

9. CALDICOTT GUARDIAN

- In 1997 the Caldicott Report (named after the author **Dame Fiona Caldicott**) was produced, which identified weaknesses in the way parts of the NHS handled confidential patient data.
- The report made several recommendations, one of which was the appointment of a **Caldicott Guardian, a senior member of staff** with a responsibility to ensure patient data is kept secure.
- Each NHS organization has to appoint a Caldicott Guardian to fulfil this role. The six key principles of the Caldicott report are:
 - *Justify the purpose(s) for using the confidential information.*
 - *Only use it when absolutely necessary.*
 - *Use the minimum that is required.*
 - *Access should be on a strict need-to-know basis.*
 - *Everyone must understand his or her responsibilities.*
 - *Understand and comply with the law.*

10. DATA PROTECTION ACT 1998

- The Data Protection Act defines UK law on the processing of data on identifiable, living people.
- It gives every living person, or their representative, the right to apply for access to their health records.
- There are eight key principles that must be complied with when processing personal data:
 - *Personal data should be processed fairly and lawfully.*
 - *Data should only be obtained for specified purposes and should not be further processed in a manner incompatible with these purposes.*
 - *Personal data should be adequate, relevant and not excessive in relation to the purposes for which they were collected.*
 - *Personal data should be accurate and where necessary kept up to date.*
 - *Personal data should not be kept longer than is needed for its intended purpose.*
 - *Personal data should be processed in accordance with the rights of the individual which the information concerns.*

- *Appropriate measures should be taken against unauthorized or unlawful processing or destruction of personal data.*
- *Personal data should not be transferred outside the European Economic Area.*
- Applications for access to health records by the patient, or their representative, must be made **in writing or electronically to the Records Manager at the hospital, with the patient's signature.**
- A fee may be charged for the release of the information. Requests should be dealt with promptly, **within 21 days and no later than 40 days** after the request has been made.
- Access may be denied, or limited, where the information might cause serious harm to the physical or mental health, or condition of the patient, or any other person, or where giving access would disclose information relating to or provided by a third person who had not consented to the disclosure.

11. FREEDOM OF INFORMATION ACT 2000

- The Freedom of Information Act deals with access to official information and **gives individuals or organisations the right to request information from any public authority.**
- **Part I of the Act**
 - Anyone can make a request for information to any public authority providing it is **in writing, states the name and address of the enquirer, and describes the information requested.**
 - The authority has the duty to confirm or deny whether it holds the information, and if it does so, to supply it **within 20 working days from receipt of request.**
 - Authorities are not obliged to provide information where they cannot find it without assistance.
- **Part II of the Act**
 - Sets out exemptions where the right of access to information is not allowed or restricted.
 - These relate to issues such as *national security, law enforcement, commercial interests, and data protection.*
- Requests from someone about their personal information are dealt with under the **Data Protection Act (See Above).**

Confidentiality source: GMC

http://www.gmc-uk.org/guidance/ethical_guidance

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II. CONSENT

• INTRODUCTION

- Consent is required for every examination, treatment, or intervention performed on a patient. **Consent may be explicit or implied.**
- **Explicit consent** is when a patient actively agrees, either verbally or in writing.
- **Implied consent** is signalled by the behaviour of an informed patient; for example, putting their arm out for a blood test.
- There are exceptions where consent is not required, such as emergency treatment and where the law prescribes otherwise (e.g. mental health law).
- There are only a few situations where written consent is legally required (e.g. the storage and use of gametes and embryos).
- **Verbal consent is otherwise as valid as written consent.**
- Consent forms do not prove valid consent they just provide some evidence that consent was obtained. Discussion with a patient regarding **consent should be documented in the notes** and state the purpose of the treatment, risks, benefits, and alternatives.

• THE KEY PRINCIPLES FOR VALID CONSENT ARE:

- *The patient must be competent.*
- *The patient must be sufficiently informed to make a choice.*
- *Consent must be given voluntarily.*

• The GMC provides guidance on the type of information doctors should provide when gaining consent. This information includes:

- The purpose of the investigation or treatment.
- Details and uncertainties of the diagnosis.
- Options for treatment including the option not to treat.
- Explanation of the likely benefits and probabilities of success for each option.
- The risks such as known possible side effects, complications, and adverse outcomes, including where intervention or treatment may fail to improve a condition.
- The name of the doctor with overall responsibility.
- A reminder that the patient can change their mind.

• WHO CAN GIVE CONSENT?

- The only person who can consent for a competent adult is **the patient themselves.**
- **A young person of any age** can consent to treatment provided they are considered to be competent (**Gillick competent**) to make the decision.
- **At the age of 16** there is a presumption that the patient is able to give valid consent.
- However, **up to the age of 18 in England, Wales, and Northern Ireland, and age 16 in Scotland**, if the person is felt to lack capacity, a person with **parental responsibility** can give consent on behalf of the patient.
- **A Lasting Power of Attorney** can consent on behalf of an adult patient once capacity is lost.

1. REFUSAL OF CONSENT

- Competent adult patients are entitled to refuse consent to treatment, even if doing so may result in permanent physical injury or death. The exception to this is where compulsory treatment is authorized by mental health legislation.
- Where the consequences of refusal are grave, it is important that the patient understands this.
- Doctors must respect a refusal of treatment if the patient is a competent adult, who is properly informed, and not being coerced.
- In England, Wales, and Northern Ireland, refusal of treatment by competent under-18s is not necessarily binding upon the doctors.
- *The courts have ruled that patients under 18 have a right to consent to treatment, but not to refuse it if this would put their health in serious jeopardy.*
- In such circumstances consent may be gained from an **adult with parental responsibility or a court**.
- *In Scotland, it is likely that **neither parents nor the courts** are entitled to override a competent young patient's decision, although this has not been tested in the courts.*
- Cases of refused consent are best discussed with senior medical staff, the hospital legal department, and/or medical defence societies.

2. CONSENT FOR EMERGENCY TREATMENT

- Consent should be sought for emergency treatment if the patient is competent.
- If consent cannot be obtained, medical treatment that is **in the patient's best interest**, and is immediately necessary to save life or avoid significant deterioration in the patient's health, should be provided.
- If the patient has appointed a **welfare attorney**, or there is a court-appointed deputy or guardian, this person, where practicable, must be consulted about treatment decisions.
- If the patient is **under 18 years old in England, Wales, and Northern Ireland, or under 16 in Scotland**, and unable to give consent due to lack of capacity or illness, **anyone with parental responsibility can provide consent**.
- If treatment is urgent and nobody with parental responsibility is available, treatment can proceed, without consent, **provided it is in the patient's best interest**.

3. REQUESTS FOR TREATMENT

- This case concerned a wide range of issues, most of which related to decision-making at the end of life.
- However, for the purposes of this guidance, the key point is the Court of Appeal's opinion that doctors are **under no legal or ethical obligation to agree to a patient's request** for treatment if they consider the treatment is **not in the patient's best interests**.

4. GILICK COMPETENCE AND FRASER GUIDELINES

- Gillick competency and Fraser guidelines refer to a legal case which looked specifically at whether doctors should be able to give contraceptive advice or treatment to under 16-year-olds without parental consent.

- But since then, they have been more widely used to help assess whether a child has the maturity to make their own decisions and to understand the implications of those decisions.
- **The Fraser guidelines** refer to the guidelines set out by Lord Fraser in his judgment of the **Gillick** case in the House of Lords (1985), which apply specifically to contraceptive advice.
- Lord Fraser stated that a Doctor could proceed to give advice and treatment to a young person under the age of 16 if:
 - She had sufficient maturity and intelligence to understand the nature and implications of the proposed treatment,
 - She could not be persuaded to tell her parents or to allow her doctor to tell them,
 - She was very likely to begin or continue having sexual intercourse with or without contraceptive treatment,
 - Her physical or mental health were likely to suffer unless she received the advice or treatment,
 - The advice or treatment was in the young person's best interests.
- This case was specifically about contraceptive advice and treatment, but the case of *Axon, R (on the application of) v Secretary of State for Health [2006] EWHC 37 (Admin)* makes clear that the principles apply to decisions about treatment and care for sexually transmitted infections and abortion, too.
- As a result of this decision, a young person under 16 with capacity to make any relevant decision is often referred to as being '**Gillick competent**'.

Consent and Capacity source: GMC

http://www.gmcuk.org/guidance/ethical_guidance/consent_guidance_common_law.asp

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III. MENTAL CAPACITY

1. ASSESSING CAPACITY

- The Mental Capacity Act aims to protect people who lack capacity, and maximize their ability to make decisions. The Act came into full force in October 2007.
- The Act is underpinned by five statutory principles:
 - *A person should be assumed to have capacity unless it is established that they lack capacity*
 - *A person should not be treated as lacking capacity unless all practical steps have been tried to enable capacity.*
 - *A person is allowed to make an unwise decision.*
 - *If a person lacks capacity then decisions should be made in their best interests.*
 - *Any decision made should be the least restrictive option.*
- Patients should always be assumed to have capacity but if there is reason to believe a patient lacks capacity it should be assessed using the two-stage test:
 - *Does the person have an impairment, or disturbance of the functioning, of their mind or brain?*
 - *Does the impairment or disturbance mean that the person is unable to make a specific decision when they need to?*
- **A person lacks capacity** if they are unable to (any three will score a mark each): **"URUC"**
 - *Understand the information relevant to the decision*
 - *Retain the information*
 - *Use or weigh the information*
 - *Communicate the decision (by any means)*

2. ADVANCE DIRECTIVES AND CAPACITY

- **WHAT IS AN ADVANCE DECISION?**
 - An advanced decision (**'living will'**) allows an adult (over 18 years) with capacity to state how they wish to be treated if they suffer a loss of capacity.
 - Advanced decisions usually relate to the refusal of medical treatment but can be statements authorizing or requesting certain procedures or treatments.
 - Advanced refusals of treatment are legally binding; however, advanced request or authorizations are not, but should be taken into account when assessing best interests.
 - Valid and applicable advance decision to refuse treatment must be specific to the treatment in question.
 - It has the same force as a contemporaneous decision.
 - A Lasting Power of Attorney appointed before the advanced decision cannot overrule it, nor can the Court of Protection.
 - The refused treatments **must all be named in the advance decision.**
 - Patient may want to refuse a treatment in some situations, but not others. If this is the case, he/she needs to be clear about all the circumstances in which he/she wants to refuse this treatment. Patient can refuse a treatment that could potentially keep him/her alive (known as life-sustaining treatment). This includes treatments such as **ventilation and cardio-pulmonary resuscitation (CPR).**
 - An advance decision is not the same as an **advance statement.**
 - Deciding to refuse a treatment is not the same as asking someone to end life or to help end your life; **Euthanasia and assisted suicide** are illegal under English law.

- **WHO MAKES AN ADVANCE DECISION?**

- Patient makes the advance decision, as long as he/she has the mental capacity to make such decisions.
- Patient may want to make an advance decision with the support of a clinician.
- A decision to refuse life-sustaining treatment in the future needs to be:
 - *Written down*
 - *Signed by the Patient*
 - *Signed by a witness*
- If Patient wishes to refuse life-sustaining treatments in circumstances where he/she might die as a result, he/she needs to state this clearly in the advance decision.

- **IS AN ADVANCE DECISION LEGALLY BINDING?**

- Yes it is, as long as it:
 - *Complies with the **Mental Capacity Act***
 - *Is valid*
 - *Applies to the situation*
- If the advance decision is binding, it takes the place of decisions made in the patient's best interest by other people.

- **AN ADVANCE DECISION MAY ONLY BE CONSIDERED VALID IF:**

- *The advanced decision must have been made by the patient when they were an adult (over 18), had capacity, and were properly informed.*
- *The statement should specify precisely what treatment is to be refused and the circumstances in which the refusals should apply.*
- *The advanced decision will only apply once the patient lacks capacity to consent to or refuse treatment.*
- *An advanced decision that relates to the refusal of life-sustaining treatments must be written, signed, and witnessed. The patient must acknowledge in the written decision that they intend to refuse treatment, even though this puts their life at risk.*

- **AN ADVANCED DECISION MAY BE INVALID IF:**

- The decision was withdrawn while the person had capacity.
- After the advance decision was made, a Lasting Power of Attorney was appointed and given express authority to make the treatment decisions covered by the advanced decision.
- The person has done something that clearly goes against the advanced decision, which suggests they have changed their mind.
- If the possibility of an advanced decision is raised for a patient who currently lacks capacity, reasonable efforts must be made to find out the details of the decision.
- This may involve contacting the **patient's GP, looking at the hospital medical notes, and discussions with the patient's relatives**. If emergency treatment is required, this should not be delayed to look for an advanced decision **if there is no indication that one exists**.
- **If there is an indication that one exists**, the validity and applicability should be assessed and the decision adhered to, if valid.
- If the advanced decision is not valid or applicable, the treatment given **should be in the patient's best interest**.
- *Advanced decisions can be overruled if the patient is being treated compulsorily under mental health legislation. However, a valid and applicable advanced refusal of treatment for conditions that are not covered by the compulsory powers of the legislation must be adhered to.*

- **HOW DOES AN ADVANCE DECISION HELP?**

- As long as it is valid and applies to the situation, an advance decision gives the health and social care team clinical and legal instructions about the patient's treatment choices.
- An advance decision will only be used if, at some time in the future, the patient is not able to make his/her own decisions about the treatment.

- **DOES IT NEED TO BE SIGNED AND WITNESSED?**

- **Yes it does**, if chosen to refuse life-sustaining treatment – in which case, the advance decision must be written down, and both the patient and a witness must sign it.
- Patient must also include a statement that the advance decision applies even if his/her life is at risk.

- **WHO SHOULD SEE IT?**

- Patient has the final say on who sees it, but he/she should make sure that the family, carers, or health and social care professionals know about it, and know where to find it.
- A copy can be kept in the medical records.

3. LASTING POWER OF ATTORNEY

- The Mental Capacity Act allows people over 18 years of age, who have capacity, to appoint a Lasting Power of Attorney (LPA).
- The person making the LPA is referred to as the '**Donor**'.
- A LPA can be appointed to make decisions on health and personal welfare, and/or property and financial affairs on behalf of the donor should they lose capacity in the future.
- The LPA is bound by the principles set out in the Mental Capacity Act and must make decisions in the donor's best interest.
- **A valid LPA requires** a signed certificate completed by an independent third party, which confirms that the donor understands the scope and purpose of the LPA and was not put under any pressure to make the LPA.
- The LPA must be registered with the Office of the Public Guardian.
- A personal welfare LPA can make healthcare decisions for the donor once they lack capacity and can consent on their behalf to treatment and social care decisions.
- There are specific situations when the LPA cannot consent to or refuse treatment:
 - *When the donor has capacity to consent.*
 - *When the donor has made an advanced decision to refuse treatment (unless the LPA was appointed after the advanced decision and the donor gave permission to the LPA to refuse treatment).*
 - *When the decision relates to life-sustaining treatment and this has not been expressly authorised in the LPA.*
 - *When the donor is detained under the Mental Health Act.*
- An LPA does not have the power to demand specific treatments if they are not felt to be necessary or appropriate. All LPAs are registered with the Office of the Public Guardian, who can confirm whether a patient has a LPA or not.
- If the medical team and LPA disagree on the best treatment for the patient, the case can be referred **to the Court of Protection**.
- Whilst a decision is reached the patient can be treated to prevent serious deterioration.

4. COURT OF PROTECTION

- The role of the Court of Protection is to **protect individuals who lack capacity and make difficult decisions about their care and welfare.**
- The Court of Protection can:
 - *Determine whether an LPA is valid or not.*
 - *Give directions about using an LPA.*
 - *Remove an LPA.*
 - *Settle disputes over healthcare and treatment of a person lacking capacity.*

5. INDEPENDENT MEDICAL CAPACITY ADVOCATES (IMCA)

- The role of an **IMCA** is to **support and represent a person who lacks capacity** in making a specific decision, who has no-one (other than paid carers) to support them.
- The IMCA:
 - *Provides support for the person who lacks capacity.*
 - *Represents the person without capacity in discussions about proposed treatment.*
 - *Provides information to work out what is in a person's best interest.*
 - *Questions or challenges decisions that they believe are not in the best interests of the person lacking capacity.*
 - *Presents individuals' views and interests to the decision-maker.*
- **The IMCA is not the decision-maker and cannot consent on behalf of the person** but the information and views expressed by the IMCA must be taken into account.
- An IMCA must be involved in decisions relating to providing, withholding, or stopping serious medical treatment. In an emergency situation, it is unlikely that there is time to instruct an IMCA so the patient should be treated according to **best interest principles and any decisions clearly documented.**
- If the IMCA disagrees with the proposed treatment and further discussion does not resolve this then the IMCA may use the formal complaints system to settle the case, or in more urgent cases, refer to the Court of Protection for a decision.

6. BEST INTERESTS

- The Mental Capacity Act states that any act done or decision made on behalf of a person who lacks capacity must be in their best interests.
- The Act sets out the factors that should be considered when deciding what is in a person's best interests:
 - *Past and present wishes and feelings.*
 - *Beliefs and values that may have influenced the decision being made, if the person had capacity.*
 - *Other factors the patient would be likely to consider if they had capacity.*
- In trying to assess the person's best interests you should:
 - *Encourage the person who lacks capacity to participate in the decision.*
 - *Avoid discrimination.*
 - *Try to identify all the issues most relevant to the person and to the decision being made.*
 - *If possible, defer the decision if the patient is likely to regain capacity.*

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